No. 95173-0

IN THE SUPREME COURT OF THE STATE OF WASHINGTON

L.M., a minor, by and through his Guardian ad Litem
WILLIAM L.E. DUSSAULT,

Plaintiffs/Petitioners,

vs.

LAURA HAMILTON, individually and her marital community; LAURA HAMILTON LICENSED MIDWIFE, a Washington business,

Defendants/Respondents.

AMICUS CURIAE BRIEF OF DR. MICHAEL D. FREEMAN

David S. Heller
WSBA No. 12669
860 SW 143rd Street
Seattle, WA 98166
(206) 243-7300

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I. IDENTITY & INTEREST OF AMICUS CURIAE

Dr. Michael D. Freeman, MedDr, PhD, MPH, FAAFS is, among other things, a doctor of medicine and an epidemiologist, an associate professor of forensic medicine at Maastricht University Faculty of Health, Medicine, and Life Science, an affiliate professor at Oregon Health & Science University School of Medicine, and a US Fulbright Fellow. A copy of his CV is attached as Appendix A. Briefly, Dr. Freeman practices in the fields of forensic medicine and forensic epidemiology and has published approximately 190 peer-reviewed scientific papers, abstracts, book chapters, and books upon topics that include injury causation and injury biomechanics, including one of the largest ever published studies of the causes of brachial plexus birth injuries.

Dr. Freeman has an interest in preventing the misuse and misapplication of science, and of biomechanical testimony in particular, in our trial courts. Dr. Freeman, through the undersigned counsel, is filing contemporaneously with this brief a motion for leave to file an amicus curiae brief in this matter, pursuant to RAP 10.1(e), 10.6(a) and (b).

II. STATEMENT OF THE CASE & INTRODUCTION

The facts are set forth in the Court of Appeals opinion herein, and in the briefs of the parties. It apparently is undisputed that defendant midwife Hamilton delivered LM and that LM has a permanent injury to

all five nerves of his brachial plexus, including avulsions (nerves torn away from the bone). Testimony of Dr. Howard Mandel, MD, 10/21/15 RP 66, ll. 15-25.

An injury may have more than one proximate cause. If the defendant's negligence was a proximate cause of the injury, the defendant is liable for the harm even though other contributing causes may also have existed. WPI 15.04; *Brashear v. Puget Sound Power & Light Co., Inc.*, 100 Wn.2d 204, 667 P.2d 78 (1983). The legal liability issue in this case therefore is whether one of the proximate causes of LM's brachial plexus avulsions and other injuries was negligence by the defendant.

The defense claimed that LM's mother herself caused LM's injuries by pushing during labor – the so-called "Natural Forces of Labor" defense. In support of that defense, defendant called as a witness biomechanical engineer Prof. Allan Tencer. 10/27/15 RP 4-39.

III. ISSUE PRESENTED

Should a biomechanical engineer's opinion about the cause of an injury be admitted into evidence where he did not follow generally accepted methodologies in arriving at that opinion, where the opinion is contrary to established medical literature, and where the opinion itself is not generally accepted in the relevant scientific community?

IV. ARGUMENT

A. Professor Tencer's opinion that natural forces of labor could cause LM's injuries was not reached by methodology which is generally

accepted in the relevant scientific community, and his opinion should not have been admitted under the *Frye*[1] test.

1. Frye applies to this case and mandates exclusion of Prof. Tencer's opinions unless he used generally accepted methodology to reach those opinions.

The application of *Frye* to issues of medical causation was addressed by this Court in *Anderson v. Akzo Nobel Coatings, Inc.*, 172 Wn.2d 593, 603, 260 P.3d 857 (2011):

> The primary goal [under *Frye*] is to determine "whether the evidence offered is **based on established scientific methodology**." Both the scientific theory underlying the evidence and the technique or methodology used to implement it must be generally accepted in the scientific community for evidence to be admissible under *Frye*. [citations omitted, emphasis added].

Anderson recognized, at 611, a distinction between the method used to reach the witness's opinion, which must pass the *Frye* test, and the witness's opinion itself, which in some cases need not pass *Frye*. But neither Prof. Tencer's methodology in reaching his opinion, nor his opinion itself, passes *Frye*, and *Anderson* therefore does not support admitting his testimony.

Even if *Anderson* does at first blush appear to support admission of Prof. Tencer's testimony, it is in fact distinguishable on several grounds pertaining to Prof. Tencer's credentials and to the relationship between his opinions and the relevant scientific literature.

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[1] *Frye v. United States*, 54 App. D.C. 46, 293 F. 1013 (1923).

In *Anderson*, plaintiff alleged brain damage due to exposure to toxic solvents. The medical literature reported that brain damage could be caused by these solvents, but was silent about whether these solvents could cause plaintiff's particular type of brain damage. This Court nevertheless permitted a medical doctor to testify that these solvents had caused plaintiff's damage.

The proffered testimony in *Anderson* came from a medical doctor, Dr. Sohail Khattak, and not from an engineer such as Prof. Tencer. *Anderson*, at 598.[2] Dr. Khattak testified to a reasonable degree of medical certainty. These facts distinguish *Anderson* from the instant case.

Dr. Khattak also undertook the necessary differential diagnosis, considering and excluding other plausible causes of plaintiff's brain damage as unlikely. *Anderson* at 610. Differential diagnosis and the consideration of other plausible causes is part of the generally accepted methodology of determining medical causation. Dr. Khattak acknowledged that the type of injury plaintiff had was found in 1 in 2,500 babies who had no known exposure to toxic solvents. Obviously, some cause other than toxic exposure was a possible cause of that plaintiff's injury, but

[2]We are aware that this Court held in *Frausto v. Yakima HMA, LLC*, 188 Wn.2d 227, 229, 393 P.3d 776 (2017) that a non-doctor, an ARNP, could testify to the causation of an injury. But ARNPs are independently licensed health care practitioners who are legislatively empowered to diagnose injuries, pursuant to RCW 18.79.050. Prof. Tencer has no such credentials or license.

was so rare that it was not a probable cause. Prof. Tencer did not offer this kind of analysis.

In *Anderson* the medical literature was silent on the issue of whether plaintiff's type of injury was caused by toxic solvents. Society, and the law, cannot wait forever for science to answer a specific question that it might never get around to addressing. So as a matter of sound public policy, the Court might permit medical doctors to fill in the gaps, with a professional opinion based upon education, training and experience. But this policy consideration does not apply in the instant case, because Prof. Tencer is not a doctor, and, as will be explained below, because the medical literature is not silent on the cause of brachial plexus avulsion. As will be discussed *infra* at pp. 18-20, the literature contradicts Prof. Tencer's claim that maternal labor alone can cause such injuries.

2. In science, the words "hypothesis" and "theory" mean different things and cannot be used interchangeably.

In the discussion which follows, it is important to note the distinction science makes between a hypothesis and a theory. These words are terms of art, and their meanings are not identical to their common dictionary meanings. *See, e.g.*, Federal Judicial Center, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 50-51 (3d. ed. 2011).

A hypothesis is at best an educated guess, which has been proposed so that it can be properly tested through experiment to determine

whether it is correct or incorrect. Without testing, a hypothesis is no better than speculation, and it is not science. On the other hand, a theory is a principle or a set of principles which may have started out as a hypothesis but which has been thoroughly tested. For example, one of the most thoroughly tested and validated, and therefore one of the most universally accepted sets of principles in all of physics, is Albert Einstein's Special Theory of Relativity. *Id.*

> *3. Epidemiology[3] and differential diagnosis, not engineering calculations, are the appropriate scientific mechanism for determining the cause of a specific type of injury or illness. Cause is always assessed by examining the likely contribution of a suspected cause versus plausible competing causes; it is never determined merely by demonstrating that a particular competing cause is theoretically possible or plausible.*

The generally accepted way to determine the cause of an injury is to form a hypothesis about the cause, observe the manifold plausible causes in the real world, and study them to confirm or refute the hypothesis. This study is called epidemiology.

How science test a hypothesis about the cause of a type of injury? We obviously cannot perform injury-risking experiments upon laboring mothers and their babies. So part of the generally accepted procedure is

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[3]Epidemiology is "the science concerned with the study of the factors determining and influencing the frequency and distribution of disease, injury, and other health-related events..." ENCYCLOPEDIA AND DICTIONARY OF MEDICINE, NURSING, & ALLIED HEALTH (7th ed.) 2003. The use of epidemiological methods as a correct way of determining the cause of a medical condition was tacitly acknowledged in *Anderson v. Akzo Nobel Coatings, Inc.*, *supra*, at 603-04, 611-12 (2011).

to use epidemiological studies to identify factors associated with an increased or decreased risk of injury.

An early example illustrates the point. Dr. Ignác Semmelweis was a 19th-century Hungarian obstetrician. He became one of the pioneers of epidemiology when, in 1847, he published a book entitled ETIOLOGY, CONCEPT, AND PROPHYLAXIS OF CHILDBED FEVER.[4] Semmelweis observed that women who delivered in hospital wards where they were attended by medical doctors developed childbed fever at eight times the rate of women who delivered in wards attended by midwives.

Semmelweis' search for causation focused on behavioral differences between obstetricians and midwives. It was common for obstetricians to spend their early mornings performing autopsies upon the corpses of women who had died in childbirth; then the doctors made their rounds in the wards. Midwives, being viewed in those days as "mere women", were not permitted to participate in autopsies. Semmelweis hypothesized that somehow the doctors were carrying illness from the dead, autopsied women to the living patients.

He conducted a proper scientific study. He formulated a hypothesis, that certain healthcare providers were causing illness, based upon the observed difference in infection rates between doctors and

[4]Childbed fever is a type of potentially fatal post-partum infection which in Semmelweis' time was the single most common cause of maternal death.

midwives. He further hypothesized that the different infection rates could be explained by the difference in how the two groups spent their mornings.

He then confirmed his hypotheses through an experiment. He recorded the rate of childbed fever in the wards, then implemented a new protocol, then recorded the new rate of childbed fever. Semmelweis' new protocol was simple: he ordered the doctors under his supervision to wash their hands after autopsies and before examining patients.[5] The results were dramatic. The rate of childbed fever fell from 18% to 3%.

Some of Semmelweis' contemporaries accepted his findings, and many lives were saved. But others did not want to acknowledge that they might be harming their patients, so they loudly rejected Semmelweis' discovery. They refused to wash their hands. They attributed the death rate differential to "divine providence"[6], or they claimed that the mothers fell ill for "hysterical" reasons when male doctors examined them.[7] Of course, Semmelweis' work ultimately was vindicated when later scientists developed modern germ and hygiene theory.

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[5]Semmelweis lived in the time before the development of modern germ theory.

[6]"I prefer to believe that childbed fever is brought about by the will of Providence, which I understand, than that it is caused by an unknown contagion, which I don't." Dr. Charles D. Meigs, *On the nature, signs, and treatment of childbed fevers* 104 (1854).

[7]Medical doctors in the 1800's believed that a person who experienced emotional distress could develop a life-threatening condition called "brain fever." Supposed symptoms included a high temperature, weakness leading to being bedridden, and temporary insanity. *See, e.g.*, Dr. A. C. Doyle, *The Naval Treaty* (1893)

The work of Dr. Semmelweis provides an analogy to brachial plexus injury, because of his correct use of epidemiology and because he studied the increased risk of injuries depending upon the profession of the practitioner attending the delivery. The medical literature establishes that:

1. Shoulder dystocia "occurs when the anterior shoulder of the fetus becomes lodged behind the superior symphysis pubis, preventing further delivery." MCGRAW-HILL CONCISE DICTIONARY OF MODERN MEDICINE (2002) (emphasis added).

2. Shoulder dystocias attended by a midwife, nurse, corpsman, or osteopath are at 3.1 to 4.9 times increased risk of neonatal brachial plexus injury, versus dystocias attended by an MD-OB. Lynne V. McFarland, PhC, et al, *Erb/Duchenne's Palsy: A Consequence of Fetal Macrosomia and Method of Delivery*, 68 OBSTETRICS & GYNECOLOGY 786 (1986).[8]

Similar to what Semmelweis recognized so long ago, a plausible hypothesis for the higher rate of brachial plexus injury to babies delivered by certain types of healthcare providers is that those types of health care providers can cause, or contribute to causing, brachial plexus injury.

An example of the generally accepted methodology for determining medical causation among competing plausible causes, and of the role of differential diagnosis therein, was described in *Etherton v. Owners Insurance Co.*, 829 F.3d 1209, 1218-19, 1223 (10th Cir. 2016). This process requires, among other steps, that one consider other potential causes of an injury or illness. The *Etherton* court approved this method in

[8]Erb/Duchenne's Palsy is a type of brachial plexus injury. Fetal macrosomia is medical jargon for "large baby". A copy of Dr. McFarland's paper is attached as Appendix B.

light of *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579, 113 S.Ct. 2786, 125 L.Ed.2d 469 (1993) and *Daubert*'s progeny.[9]

4. Prof. Tencer failed to follow generally accepted scientific procedures by using a hypothesized, unproven cause of injury as if it were an accepted, proven, most probable cause.

Prof. Tencer hypothesizes that natural forces of labor caused LM's brachial plexus injuries, including avulsions. But a hypothesis is not a tested theory, and it cannot be treated as anything other than speculation unless it has been tested and proved.

Prof. Tencer developed his hypothesis about the cause of LM's injury via abductive reasoning. Abductive reasoning is reasoning based upon limited observation, and it can be used to generate a possible explanatory hypothesis. It was perhaps most famously used by the fictional detective Sherlock Holmes, who observed characteristics about a person and then hypothesized about that person's profession or life story.[10]

In the real world of science and medicine, hypotheses always require proof before being accepted. Abductive reasoning is suitable for developing a hypothesis about a plausible cause for an injury. But it is not suitable for testing or confirming that hypothesis. It cannot be used to

[9]Of course, in matters of scientific evidence, Washington has followed *Frye*, not *Daubert*, though the outcome of the two analyses frequently is the same. *See, e.g., Reese v. Stroh*, 128 Wn.2d 300, 907 P.2d 282 (1995).

[10]Holmes called it "deductive reasoning" but this was largely a misnomer.

quantify whether that hypothetical cause is a substantial factor, or the most probable factor, or not a factor at all, in causing the injury.

As explained at p. 6, *supra*, the correct, generally accepted procedure is to identify the multiple factors which could cause or contribute to causing the injury. As verified by the medical literature, it is generally accepted that there is more than one factor which may contribute to causing brachial plexus injury. The literature documents that the risk of brachial plexus injury is increased for delivering practitioners in certain professions. *See*, McFarland, cited *supra*, at p. 9. Other established risk factors include the use of forceps in delivery (injury 4.6 times more likely to occur), and the use of vacuum extraction in delivery (injury 2.3 times more likely to occur); both of these activities involve the practitioner exerting traction (pulling) on the baby.[11]

Once a set of risk factors for a type of injury is established, it is generally accepted that the next step is to weigh those factors against one another. Statistics, probability, and/or differential diagnosis are used to determine the most probable cause or causes of an injury. In order to show, as Prof. Tencer claimed or implied, that the cause of a brachial plexus injury was maternal labor, it would be scientifically necessary to rule out other likely causes, or at least to establish that other causes were less likely

[11] *See, e.g.*, Michael D. Freeman, et al, *A multistate population-based analysis of linked maternal and neonatal discharge records to identify risk factors for neonatal brachial plexus injury*, 136 INT. J. GYNECOL. OBSTET. 331-36 (2017) (attached in Appendix C).

than maternal labor. Prof. Tencer did neither, nor did he show that it had been done by others. When a witness fails to follow proper methodology in coming to a conclusion, the conclusion is unreliable. *Lakey v. Puget Sound Energy, Inc.*, 176 Wn.2d 909, 920, 296 P.3d 860 (2013).

In fact, the existing scientific/epidemiological data regarding brachial plexus injuries makes it extremely unlikely that LM's injury could have been caused by maternal labor alone. All vaginal births involve forces of maternal labor, yet most vaginal births do not result in brachial plexus injuries, let alone brachial plexus avulsion. All vaginal births which involve shoulder dystocia also involve maternal labor, yet all vaginal births involving shoulder dystocia do not result in brachial plexus injuries, let alone brachial plexus avulsion. Obviously, something more than merely giving birth must cause or contribute to causing brachial plexus avulsion. The above data regarding practitioners, forceps, and vacuum extraction identify some of those potential contributing causes.

It is not simply Prof Tencer's hypothesis which is not generally accepted. The use of a hypothesis generated in this way and left untested also is not generally accepted. And the use of an untested hypothesis which contradicts the published medical literature is not generally accepted.

A hypothesis with no or insufficient real world evidence to support it is speculation, not science. One might just as well hypothesize

that LM's brachial plexus injury was caused because at the time of his birth, the Moon was in its Seventh House. Anyone can proffer a hypothesis that appears to exonerate a civil defendant, but when there is no or insufficient scientific evidence to support it, and where that hypothesis is contradicted by the applicable literature, then the hypothesis is not generally accepted and should be inadmissible under *Frye*.

The established view in the medical world is that practitioner traction (pulling) is the most likely cause of a brachial plexus avulsion. In science, the burden of proof rests upon the person claiming that the established view is wrong. Unless that burden of proof is someday met through proper methods, Prof. Tencer's causation hypothesis will never achieve general acceptance. To date, it has not.

5. Prof. Tencer's methodologies and opinions in this case are scientifically flawed because they use "average" information in a manner that is not generally accepted in the scientific community.

Prof. Tencer attempted to solve his lack of specific information about the forces involved in LM's delivery by using "averages". 10/27/15 RP 15-16. An average is calculated by adding up and dividing a certain characteristic of different people or things, not one of whom may actually be "average".[12]

[12]If a certain type of house costs $140,000 when located in Omak and $750,000 when located in Seattle, one could say that the average cost for this type of house is $445,000. But that "average" figure tells us nothing useful about what a home costs in Omak or in Seattle.

Prof. Tencer's "average" numbers are themselves suspect. They were based in part upon an article, CP 2375; 10/27/15 RP 11, 26, which contained "calculations" obtained by assuming: an imaginary uterus and a baby's body, both perfect ellipsoids; an imaginary baby's head that was a perfect sphere; and a contact area between the imaginary mother's symphysis pubis and the baby that was a perfect trapezoid. CP 3200. Using these questionable numbers, Prof. Tencer speculated that "average" natural forces of labor can cause the rupture and avulsion of a baby's brachial plexus. 10/27/15 RP 22, ll. 6-9.

Applying "average" data to a specific event such as the injuries to LM is not a generally accepted method of determining the forces involved in that event. Applying "average" data to a specific injury also is not a generally accepted method of determining the cause of that injury. Different midwives will pull on a baby with different amounts of force. Some women are in labor for 2 hours and some for 2 days, a fact consistent with the common-sense recognition that the sizes and shapes of women and their birth canals, the sizes and shapes of babies, and the "pushing ability" of women in labor, all vary widely. There is no way to know where LM's mother or LM or defendant midwife fell within any alleged "average range".[13] Such testimony is inadmissible under *Frye*.

[13]As far as we can discern, the record does not show that the defense made any effort to measure how much force defendant exerted upon a baby during delivery. They did not,

This testimony also suffered from the same scientific flaws that often undermine biomechanical testimony in personal injury cases. It is reminiscent of Prof. Tencer's testimony in car crash cases, where he "imported 'average person' studies ... to speculate what the transfer of energy might have been in this case...." *Gilmore v. Jefferson County Public Transportation Benefit Area*, 190 Wn.2d 483, 506, 415 P.3d 212 (2018)(Yu, J., concurring).

In a motor vehicle crash, one can at least look up the size, weight, and structural composition of the motor vehicle(s) in question. Even so, it remains extremely difficult to determine the forces that a particular person involved in a particular crash experienced, especially where individual characteristics of the person, such as height, weight, head positioning, etc. cannot be determined with accuracy. *See, e.g., Stedman v. Cooper*, 172 Wn. App. 9, 19-21, 292 P.3d 764 (2012), and cases cited therein. How much more difficult would it be to accurately calculate the forces acting upon a specific baby, particularly where, as here, no actual measurements of LM or his mother were made?

For these reasons, and others, it is inappropriate and misleading to use "average person" studies to speculate either about how much force might have been applied to LM by his mother pushing, or by the

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for example, have defendant pull on some force-measuring apparatus in a manner similar to how she pulls on babies, or to how she pulled on LM.

defendant midwife pulling. It violates ER 702. *See*, *Gilmore*, *supra*, at 504-06 (Yu, J., concurring).

Furthermore, testimony that does not tell the jury about the effect of a particular event upon a particular person is speculative and irrelevant. *State v. Lewis*, 141 Wn. App. 367, 389, 166 P.3d 786 (2007). "Scientific evidence that does not help the trier of fact resolve any issue of fact is irrelevant and does not meet the requirements of ER 702." *State v. Greene*, 139 Wn.2d 64, 73, 984 P.2d 1024 (1999).

6. By offering speculative testimony about possible causes of injury, Prof. Tencer violated generally accepted scientific principles for determining the cause of an injury.

As discussed above, *supra* at pp. 10-11, proper scientific procedure requires that plausible causes or contributing causes of an injury be considered and ruled in or ruled out. Prof. Tencer did not do this. Even if his engineering calculations had been done correctly, and even if his hypothesis that maternal labor could contribute to causing brachial plexus avulsion were correct, his methodology still failed the *Frye* test. Ignoring obvious contributing causes in favor of a speculative, unlikely cause is contrary to generally accepted scientific methods of determining the cause of an injury or illness. The resulting opinion should be inadmissible under *Frye*.

Even if Prof. Tencer had found one possible cause, it was improper to ignore all the others, especially the most probable – practitioner traction. Even if it were possible for maternal labor to have contributed to causing LM's injuries, that would in no way preclude other contributing causes, including negligence by the defendant midwife.

For this reason, witnesses who offer an opinion on medical causation must testify in terms of probability, not mere possibility. *Miller v. Staton*, 58 Wn.2d 879, 885-86, 365 P.2d 333 (1961). Testimony about "other possible causes" of a plaintiff's injury is speculative and inadmissible. *Washington Irrigation & Development Company v. Sherman*, 106 Wn.2d 685, 724 P.2d 997 (1986). It was not helpful to the jury in deciding whether defendant's negligence was a proximate cause of LM's injury.

Colley v. Peacehealth, 177 Wn. App. 717, 312 P.3d 989 (2013) might be interpreted as permitting a defense witness to testify about speculative "other possible causes" in a medical malpractice case. But *Colley* should be distinguished and limited to its unusual facts. First, the defense witnesses in *Colley* were medical doctors, not engineers. Second, plaintiffs in *Colley* were arguing something close to a res ipsa loquitur case – they claimed there was no explanation for the plaintiff's brain damage other than oxygen deprivation, and that oxygen deprivation proved medical malpractice had occurred. *Colley*, at 729. The court therefore allowed

the defense to offer medical testimony about other possible causes of the plaintiff's brain damage, to respond to plaintiff's assertion that the existence of his brain damage alone was proof of malpractice.

Here, plaintiff was not solely relying upon a theory that his injuries were proof of malpractice. Plaintiff offered expert medical testimony, based upon review of the actual video of LM's delivery, that this particular defendant midwife pulled excessively upon LM after LM developed shoulder dystocia, and that her pulling was a proximate cause of LM's brachial plexus avulsion. CP 1640; Testimony of Dr. Howard Mandel, MD, 10/21/15 RP 70. Because plaintiff's experts did not rely upon the fact of injury alone to prove malpractice, no *Colley*–based exception to the usual rule excluding "possible" causes should apply.[14]

B. Prof. Tencer's hypothesis that the force of maternal labor acting alone can cause brachial plexus avulsion is not generally accepted in the relevant scientific community.

As explained above, Prof. Tencer did not follow generally accepted methodologies to reach his opinion. Moreover, his opinion itself, that brachial plexus injury can be caused by maternal labor alone, is not generally accepted.

Dr. Freeman has not been able to find any reliable reports in the published, peer-reviewed medical literature, of a baby who suffered

[14]In some cases, the injury alone is evidence of malpractice. In others, it is not. If the trial court perceived a risk in a particular case that jurors might wrongly infer malpractice from injury alone, it could be addressed through jury instructions such as WPI 105.07.

permanent brachial plexus injury – much less a brachial plexus avulsion – in the absence of pulling or external traction instituted by the delivering practitioner. *See also*, Testimony of Dr. Howard Mandel, MD, 10/21/15 RP 69, ll. 2-5 (no literature ever showed avulsion caused by natural forces). In short, there is no observational, real world evidence that an avulsion has ever occurred without external traction, *i.e.*, pulling by the practitioner.

Some papers may exist which hypothesize that maternal labor could be the sole cause of a permanent avulsion injury to a baby's brachial plexus. That does not mean there is a general acceptance that this can happen. *See, e.g.*, Testimony of Dr. Stephen Glass, MD, 10/22/15 RP 116, ll. 2-3 (one report deemed unreliable because case was in litigation when written); RP 118-19, ll. 2-4 (no data to support claim that forces of labor cause most if not all brachial plexus injuries).

Controversial or speculative papers are and sometimes should be published, so that the scientific community can debate, verify, or refute them. This is part of how science advances. But controversial and speculative papers do not acquire general acceptance unless their hypotheses have been tested and validated.

The above-cited sources demonstrate that the established medical literature contradicts Prof. Tencer's testimony. When this fact is combined with the scientific flaws in Prof. Tencer's methods and testimony, and with

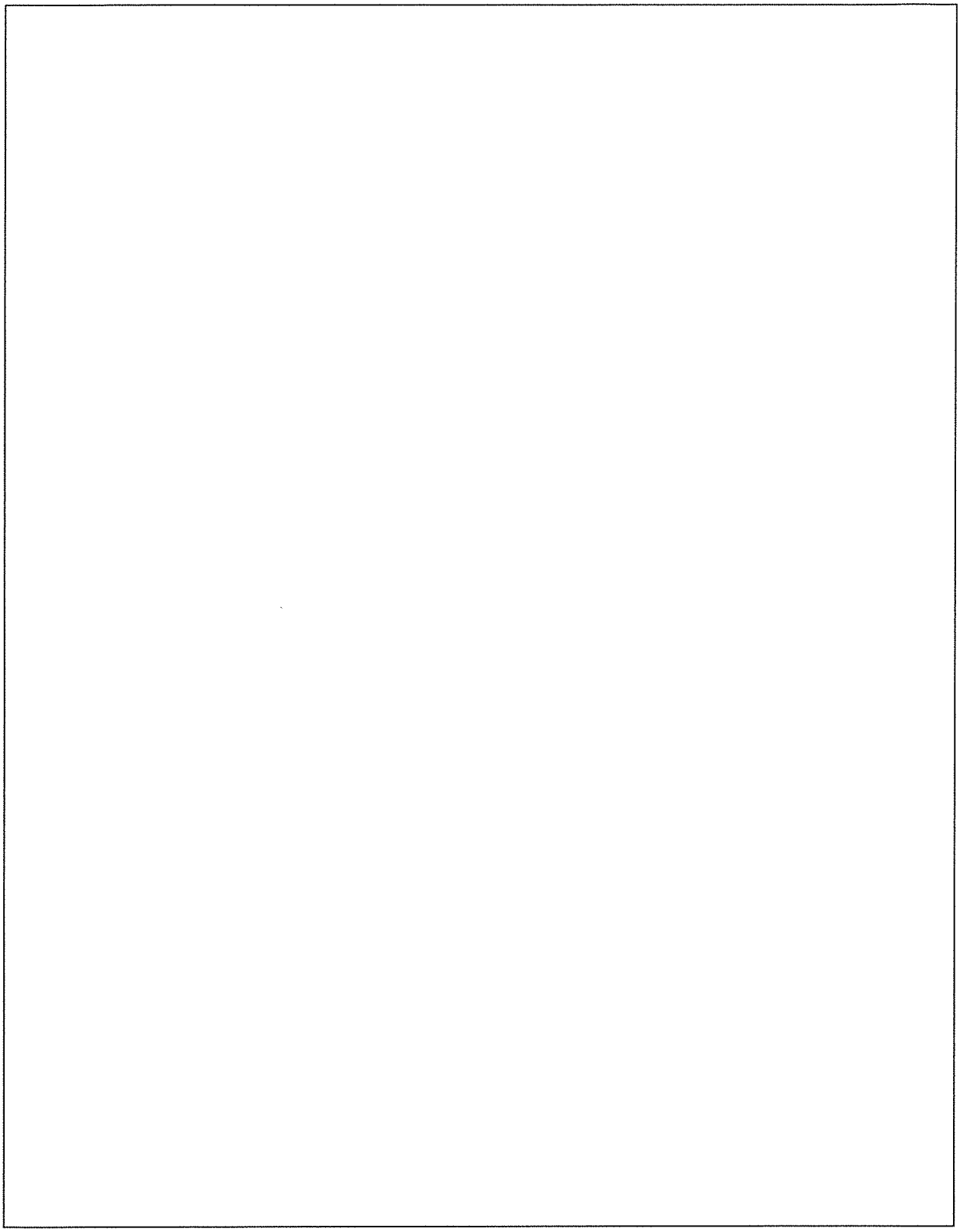
the extreme rarity or perhaps even non-existence of any known case where a baby suffered a permanent brachial plexus avulsion injury through maternal pushing alone, we can state confidently that it is **not** generally accepted in the scientific/medical community that maternal forces of labor acting alone could cause the injuries suffered by LM.

V. CONCLUSION

Prof. Tencer failed to follow generally accepted methods of developing and testing his hypothesis of the cause of injury. He failed to validate his hypothesis. He failed to consider and rule out other causes of injury. His hypothesis itself is not generally accepted, and in fact is contradicted by respected medical authority. For all these reasons, his opinions should have been excluded.

DATED this 12th day of October, 2018

David S. Heller, WSBA #12669
On behalf of Dr. Michael D. Freeman



CURRICULUM VITAE

MICHAEL D. FREEMAN

October 2018

ADDRESS
4500 Kruse Way, Plaza I, Suite 385
Lake Oswego, Oregon 97035
Tel 971-255-1008 Fax 971-255-1046
e-mail: forensictrauma@gmail.com
m.freeman@maastrichtuniversity.nl

EDUCATION

Doctor of Medicine (Med.Dr.)
Faculty of Medicine, Umeå University, Umeå, Sweden

Doctor of Philosophy (Ph.D.) Public Health/ Epidemiology
Oregon State University, Corvallis, Oregon

Master of Public Health (M.P.H.), Epidemiology/ Biostatistics
Oregon State University, Corvallis, Oregon

Doctor of Chiropractic (D.C.)
University of Western States, Portland, Oregon

Bachelor of Science (B.S.) General Science
University of Oregon, Eugene, Oregon

FELLOWSHIPS

Fulbright Specialist Roster
Bureau of Educational and Cultural Affairs and World Learning,
United States Department of State, 2017-2020 tenure

Postdoctoral Fellowship
Forensic Pathology
Section of Forensic Medicine, Department of Community Medicine and Rehabilitation, Umeå University, Umeå, Sweden 2014-2015

ACADEMIC POSITIONS

Associate Professor of Forensic Medicine – 2015-2018
CAPHRI School for Public Health and Primary Care
Maastricht University Medical Center
Maastricht, The Netherlands
Affiliate Professor of Epidemiology – 2010 to 2015
Department of Public Health and Preventive Medicine
School of Medicine, Oregon Health & Science University
Portland, Oregon
Affiliate Professor of Psychiatry – 2011 to present
Department of Psychiatry
School of Medicine, Oregon Health & Science University

Portland, Oregon
Clinical/Affiliate Associate Professor – 2005-10
Department of Public Health and Preventive Medicine
School of Medicine, Oregon Health & Science University
Portland, Oregon
Clinical Assistant Professor – 1997-2005
Department of Public Health and Preventive Medicine
School of Medicine, Oregon Health & Science University
Portland, Oregon
Adjunct Professor of Forensic Epidemiology and Traumatology – 2012-17
Department of Forensic Medicine
Faculty of Health Sciences, Aarhus University
Aarhus, Denmark
Adjunct/Honorary Associate Professor of Epidemiology and Traumatology – 2012-17
Department of Forensic Medicine
Faculty of Health Sciences, Aarhus University
Aarhus, Denmark
Adjunct Associate Professor of Forensic Medicine and Epidemiology – 2005-12
Institute of Forensic Medicine
Faculty of Health Sciences, Aarhus University
Aarhus, Denmark
Adjunct Professor – 2015 to present.
University of Western States
Portland, Oregon

EDITORIAL ACTIVITIES

Co-Editor in Chief:
Journal of Whiplash-Related Disorders 1999-2006
Associate Editor:
OA Epidemiology, 2014-present
J of Forensic Biomechanics, 2010-present
The Spine Journal 2007-present
PM&R, official scientific journal of the American Academy of Physical Medicine and Rehabilitation, 2008-present
Scandinavian Journal of Forensic Medicine, 2012-present
Editorial Board Member:
The Spine Journal 2004-present
International Research Journal of Medicine and Medical Sciences
Egyptian Journal of Forensic Sciences 2010-present
Journal of Case Reports Practice 2014-present
Austin Journal of Public Health & Epidemiology 2014-2016
Edorium Journal of Public Health 2014-present
Editorial Committee Member:
Spineline 2004-2009
Peer reviewer:
BMC Public Health
BMC Research Notes
Annals of Epidemiology (outstanding reviewer status 2015)
Orthopedics
Spine
The Spine Journal
Lancet
Mayo Clinic Proceedings
Annals of Biomechanical Engineering

Journal of the American Board of Family Medicine
Journal of Forensic and Legal Medicine
Acta Neurologica Scandanavica
Medical Science Monitor
Pain Research & Management
Journal of Back and Musculoskeletal Rehabilitation
American Society for Testing and Materials (ASTM)
Biosecurity & Bioterrorism
Annals of Medical and Health Sciences Research
Neurorehabilitation and Neural Repair
International Research Journal of Medicine and Medical Sciences
Jurimetrics
Law, Probability, and Risk
International Journal of Molecular Sciences
Journal of Rehabilitation Medicine
Arthritis
BMC Pediatrics
Journal of Back and Musculoskeletal Rehabilitation
Diagnostic and Interventional Radiology
Healthcare
Expert Review of Medical Devices
BMC Cancer

COURSES TAUGHT

PHPM 574 Forensic & Trauma Epidemiology
Department of Public Health and Preventive Medicine
Oregon Health & Science University School of Medicine
Portland, Oregon 2006-2013

Principles of Forensic Medicine and Forensic Epidemiology
Forensic Psychiatry Fellowship
Department of Psychiatry
Oregon Health & Science University School of Medicine
Portland, Oregon – 2011 to present

PHPM 503 Thesis Advising
Department of Public Health and Preventive Medicine
Oregon Health & Science University School of Medicine
Portland, Oregon 2005-present

PHPM 507 Injury and Trauma Epidemiology
Department of Public Health and Preventive Medicine
Oregon Health & Science University School of Medicine
Portland, Oregon 1999 – 2005

Forensic Epidemiology and Bioterrorism
Charles County Department of Public Health
College of Southern Maryland, Waldorf, Maryland 2014

ACTIVITIES and HONORS

Faculty, course designer and keynote speaker, "*When Science Meets Law: Forensic Epidemiology in Medicolegal Practice.*" Summer school course, Radboud Medical Center, Nijmegen, Netherlands, August 13-17, 2018.

Fulbright fellowship, US Department of State, *Forensic Epidemiology in Forensic Medicine*, March 1-15, Maastricht, Netherlands.

Keynote speaker, Gran Sesión de Epidemiología Forense. November 18, 2016 Universidad Libre, Seccional Cali, Colombia.

Vice Chair, American Academy of Forensic Sciences Standards Board Medicolegal Death Investigation Consensus Body – 2016-present
Member, American Academy of Forensic Sciences Standards Board Medicolegal Death Investigation Consensus Body – 2016-present
Affiliate Medical Examiner, Allegheny County, Pennsylvania, 2014-present
Member, Scientific Advisory Board, International Conference on Forensic Inference and Statistics. August 2014, Leiden, The Netherlands
Reviewer, National Aeronautical Space Administration (NASA) 2011
Past president, International Cellular Medicine Society, 2009 to 2012
Founding member, International Cellular Medicine Society, 2009
Member, Research Planning Committee, North American Spine Society 2007-2009
Member, Complementary Medicine Committee, North American Spine Society 2007-2009
Special Deputy Sheriff (Forensics), Vehicular Homicide Investigator, Clackamas County, Oregon, 2007-2009
Member, Crash Reconstruction and Forensic Technology (CRAFT) multidisciplinary law enforcement fatal crash investigation team, Clackamas County, Oregon, 2002-2013
Consultant Forensic Trauma Epidemiologist to the Medical Examiner Division of the Oregon Department of State Police – Occupant Kinematics, 1999-2006
Deputy Medical Examiner, Marion County, Oregon. 2000-2005
Moderator, Engineering sciences section, American Academy of Forensic Sciences 62nd Annual Meeting, Seattle, WA 2010
Co-Chair, International Whiplash Trauma Congress V, Lund, Sweden. 2011
Co-Chair, International Whiplash Trauma Congress IV, Miami, FL. October 2007.
Co-Chair, International Whiplash Trauma Congress III, Portland, OR. June 2006.
Co-Chair, International Whiplash Trauma Congress II, Breckenridge, CO. February 2005.
Co-Chair, International Whiplash Trauma Congress I, Denver, CO. October, 2003
Co-Chair, Forensic Section, International Traffic Medicine Association. Budapest, Hungary. September, 2003
Member, Blue Ribbon Panel Congressional Task Force on roller coaster-induced brain injury. Funded by a grant from the National Institute of Child Health and Human Development 2002-2003
President, Spinal Injury Foundation. Denver, CO 2002-2009
Member, Marion-Polk County C.R.A.S.H. Team - Occupant Kinematics Consultant 1999-2004
Scientific Chair, North American Whiplash Trauma Congress. Victoria, British Columbia 1999

BOARD CERTIFICATION AND ORGANIZATIONS

American Academy of Forensic Sciences, Pathology/ Biology section
Fellow (2016-present)
Member (2008-2016)
Faculty of Forensic & Legal Medicine, Royal College of Physicians, Affiliate Member
ACTAR Accredited Crash Reconstructionist, Accreditation Commission for Traffic Accident Reconstruction, Accreditation #1581
Crash Data Retrieval Technician I & II
Certification in basic and advanced crash reconstruction - Northwestern University
Diplomate, American Academy of Pain Management
Member, American College of Epidemiology
Member, Association for the Advancement of Automotive Medicine
Member, Sigma Xi Scientific Honor Society
Member, Society of Automotive Engineers
Past member, International Traffic Medicine Association
Fellow, International College of Chiropractic
Inactive member, North American Spine Society
Past member, Forensic Accident Reconstructionists of Oregon

GRANTS

2017-2020 Fulbright scholarship, Fulbright Specialist program, Bureau of Educational and Cultural Affairs and World Learning, United States Department of State.

2015 National Science Foundation Industry/University Cooperative Research Centers Program, NSF 13-594 Planning Grant: I/UCRC for Advanced Research in Forensic Science, National Center for Research on Forensic Epidemiology. Principal Investigator.

2011-2013 World Health Organization – research grant for Rwandan study of relationship between genocide and suicide and homicide victimization and offending. $50,000. Project No: AFRWA 1005685, Award No: 53975.

2010-2015 Centers for Disease Control (Administered by National University of Rwanda and OHSU) SPH/CDC $200,000 over 4 years.

2002-2003 National Institute of Child Health and Human Development – Blue Ribbon Task Force on Roller Coaster Associated Brain Injury. $75,000.

DISSERTATION SUPERVISION/MENTORING

Wendy Leith MS MPH – PhD candidate, CAPHRI School for Public Health and Primary Care, Maastricht University Medical Center (2018 to present)

Paul Nolet MPH, MSc, DC – PhD candidate, CAPHRI School for Public Health and Primary Care, Maastricht University Medical Center (2017 to present)

Huijie Wang B.Med,, M.Med. – PhD candidate, CAPHRI School for Public Health and Primary Care, Maastricht University Medical Center (2017-2018)

Dritan Bijko MD MSc – PhD candidate, CAPHRI School for Public Health and Primary Care, Maastricht University Medical Center (2017 to present)

Putri Dianita MD – PhD candidate, CAPHRI School for Public Health and Primary Care, Maastricht University Medical Center (2015 to present)

Frank Franklin Ph.D., J.D. (2013), Earle Mack School of Law, Drexel University

Bonnie Colville-Ebeling MD – PhD candidate (2012 to present) University of Copenhagen, Faculty of Health Sciences, Department of Forensic Medicine

Dimitrios Papadakis BSc, MRes, Dr.rer.nat. (2012-present) independent mentoring

Wendy Leith MS – MPH (2015) Department of Public Health & Preventive Medicine, Oregon Health & Science University School of Medicine

Konrad Dobbertin – MPH (2011) Department of Public Health & Preventive Medicine, Oregon Health & Science University School of Medicine

Apostolo Alexandridis – MPH (2011) - Department of Public Health & Preventive Medicine, Oregon Health & Science University School of Medicine

Wilson Rubanzana MD – PhD (2016) National University of Rwanda, School of Public Health, Kigali, Rwanda

Catherine Maddux-Gonzalez – MPH (2009) – Department of Public Health & Preventive Medicine, Oregon Health & Science University School of Medicine

Laura Criddle MS, RN – PhD (2008) Oregon Health & Science University School of Medicine, School of Nursing

Peter Harmer PhD – MPH (2006) Department of Public Health & Preventive Medicine, Oregon Health & Science University School of Medicine

PUBLICATIONS

Peer-reviewed journal articles

1. **Freeman MD.** Guillain-Barré Syndrome following *Vibrio parahaemolyticus*-related foodborne illness. *For Sci Med Path* (in review).

2. **Freeman MD.** Concussion risk from helmeted sports; A reexamination of data and methods. *J Forensic Biomed* 2018;9:139. doi: 10.4172/2090-2697.1000139.

3. Centeno C, Markle J, Dodson E, Stemper I, Hyzy M, Williams C, Ichim T, **Freeman MD** Symptomatic anterior cruciate ligament tears treated with percutaneous injection of autologous bone marrow concentrate: a non-controlled registry study *J Translational Med* (in press).

4. Dianita Ika Melia P, **Freeman MD**, Herkutanto H, Zeeger MP. A review of the diversity in taxonomy, definitions, scope, and roles in forensic medicine: Implications for evidence-based practice. *For Sci Med Path* 2018:doi.org/10.1007/s12024-018-0031-6.

5. Centeno C, Markle J, Dodson E, Stemper I, Hyzy M, Williams C, **Freeman MD**. Autologous Bone Marrow Concentrate and Platelet Products versus Exercise Therapy for Symptomatic Knee Osteoarthritis: A Randomized Controlled Trial (in review).

6. **Freeman MD,** Leith WM. The epidemiology of tire failure-related traffic crashes. *SAE* (in press).

7. **Freeman MD**. A practicable and systematic approach to medicolegal causation. *Orthopedics* 2018;41(2):70-2.

8. Centeno C, Markle J, Dodson E, Stemper I, Hyzy M, Williams C, **Freeman MD.** The safety and efficacy of using lumbar epidural injection of platelet lysate for treatment of radicular pain. *J Exp Orthopaedics* 2017;4:38.

9. Centeno C, Markle J, Dodson E, Stemper I, Williams C, Hyzy M, Ichim T, **Freeman MD.** Treatment of lumbar degenerative disc disease-associated radicular pain with culture-expanded autologous mesenchymal stem cells *J Translational Medicine* 2017;15:197.

10. Williams KE. **Freeman MD**. The role of the medical examiner/ coroner system in creating a public database for surveillance and information sharing on drug overdose deaths. *Academic Forensic Pathology.* 2017;7(1):60-72.

11. Leith W, Lambert W, Boenhlein J, **Freeman MD.** The association between gabapentin and suicidality in bipolar patients. *Int Clin Psychopharm* (in press).

12. Centeno C, Markle J, Dodson E, Stemper I, Williams C, Hyzy M, **Freeman MD.** Symptomatic anterior cruciate ligament tears treated with percutaneous injection of autologous bone marrow concentrate: a non-controlled prospective registry study. *BMC Musculoskeletal Disorders* (in press).

13. **Freeman MD,** Goodyear S, Leith W. Risk factors for neonatal brachial plexus injury; a multistate epidemiologic study of matched maternal and newborn discharge records. *Int J Gynecology & Obstetrics* 2017;136(3):331-336.

14. **Freeman MD, Zeegers M.** Forensic Epidemiology: An evidence-based system for analyzing individual causation in a medicolegal setting. *Austin J Public Health Epidemiol* 3(3):2016. ISSN: 2381-9014

15. Westergren H, Larson L, Carlsson A, Joud A, **Freeman MD,** Malmstrom E-M. Sex-based differences in chronic pain distribution in a cohort of patients with post-traumatic neck pain. *Disabil Rehabil* 2017 DOI: 10.1080/09638288.2017.1280543

16. Nyström A, **Freeman MD.** Central sensitization is modulated following trigger point anesthetization in patients with chronic pain following whiplash trauma. A double-blind, placebo-controlled, cross-over study. *Pain Med* 2017;0:1-6.

17. **Freeman MD**, Zeegers M. Principles and applications of forensic epidemiology in the medicolegal setting. *Law, Probability, & Risk* 2015; doi:10.1093/lpr/mgv010.

18. Centeno CJ, Al-Sayegh H, **Freeman MD** et al. A multi-center analysis of adverse events among 2,372 adult patients undergoing adult autologous stem cell therapy for orthopedic conditions. *International Orthopedics* DOI 10.1007/s00264-016-3162-y.

19. **Freeman MD**. Medicolegal causation analysis of a lumbar spine fracture following a low speed rear impact traffic crash. *J Case Rep Prac* 2015; 3(2): 23-29.

20. Uhrenholt L, **Freeman MD**, Webb A, Pedersen M, Thorup-Boel LW. Fatal subarachnoid hemorrhage associated with internal carotid artery dissection resulting from whiplash trauma. *Forens Sci Med Path* 2015;11(4):564-9.

21. Rubanzana W, Hedt-Gauthier B, Ntanganira J, **Freeman MD.** Exposure to effects of genocide as a risk factor for homicide perpetration in Rwanda: A population-based case-control study. *J Interpersonal Violence* 2015;pii: 0886260515619749. [Epub ahead of print] PubMed PMID: 26681788.

22. Centeno CJ, Al-Sayegh H, Bashir J, **Freeman MD.** A prospective study of the safety and efficacy of autologous bone marrow concentrate for the treatment of rotator cuff tears and shoulder osteoarthritis *J Pain Res* 2015;8:1-8.

23. Centeno CJ, Al-Sayegh H, Bashir J, **Freeman MD.** A dose response analysis of bone marrow concentrate injections for knee osteoarthritis. *BMC Musculoskeletal Disorders (Section: Orthopedics and biomechanics)* 2015;16:258. doi: 10.1186/s12891-015-0714-z.

24. Rubanzana W, Ntanganira J, **Freeman MD,** Hedt-Gauthier B, Risk factors for homicide victimization in post-genocide Rwanda: a population -based case- control study. *BMC Public Health* 2015;15(1):809.

25. Rubanzana W, Hedt-Gauthier B, Ntanganira J, **Freeman MD**. Exposure to genocide as a risk factor for suicide in Rwanda. *J Epidemiol Community Health* 2015 Feb;69(2):117-22.

26. Westergren H, **Freeman MD,** Malmström E-M. The whiplash enigma: still searching for answers. *Scand J Pain* 2014; http://dx.doi.org/10.1016/ j.sjpain.2014.08.003.

27. Centeno CJ, Pitts J, Al-Sayegh H, **Freeman MD.** Efficacy and Safety of Bone Marrow Concentrate for Osteoarthritis of the Hip; Treatment Registry Results for 196 Patients. *J Stem Cell Res Ther* 2014;4:242. doi: 10.4172/2157-7633.1000242

28. Centeno CJ, Pitts J, Al-Sayegh H, **Freeman MD.** Efficacy of autologous bone marrow concentrate for knee osteoarthritis with and without adipose graft. *Biomed Res Int* 2014. doi:10.1155/2014/370621

29. Centeno CJ, Pitts J, Al-Sayegh H, **Freeman MD.** Anterior cruciate ligament tears treated with percutaneous injection of autologous bone marrow nucleated cells; a pilot study. *J Pain Res* 2015;8:1–11.

30. **Freeman MD**, Cahn PJ, Franklin FA. Applied forensic epidemiology. Part 1: medical negligence. *OA Epidemiology* 2014;2(1):2.

31. Koehler S, **Freeman MD**. Forensic epidemiology; a methodology for investigating and quantifying specific causation. *Forens Sci Med Path* 2014 Jun;10(2):217-22.

32. Centeno CJ, **Freeman MD**. Percutaneous injection of autologous, culture-expanded mesenchymal stem cells into carpo-metacarpal hand joints: A case series with an untreated comparison group. *Wien Med Wochenschr* 2013:DOI 10.1007/s10354-013-0222-4

33. **Freeman MD**, Eriksson A, Leith W. Head and neck injury patterns in fatal falls: epidemiologic and biomechanical considerations. *J Forensic Legal Med* 2014;21:64-70.

34. Colville-Ebeling B, **Freeman MD**, Banner J, Lynnerup N. Autopsy practice in forensic pathology – evidence-based or experience-based? A review of autopsies performed in a case of multiple, simultaneous deaths. *J Forensic Legal Med* 2014;22:33-6.

35. **Freeman MD**, Eriksson A, Leith W. Injury pattern as an indication of seat belt failure in ejected vehicle occupants *J Forensic Sci* 2014; 59(5):1271-4.

36. Dobbertin KM, **Freeman MD**, Lambert WE, Lasarev MR, Kohles SS. The relationship between vehicle roof crush and head, neck and spine injury in rollover crashes. *Accid Anal Prev* 2013;58:46-52.

37. Centeno CJ, Schultz JR, Cheever M, **Freeman M**, Faulkner S, Robinson S. A Case Series of Percutaneous Treatment of Non-Union Fractures with Autologous, Culture Expanded, Bone Marrow Derived, Mesenchymal Stem Cells and Platelet Lysate. *J Bioengineer & Biomedical Sci S2:007* doi:10.4172/2155- 9538.S2-007

38. Woodham M, Woodham A, Skeate JG, **Freeman MD**. Long-Term Lumbar Multifidus Muscle Atrophy Changes Documented With Magnetic Resonance Imaging; A Case Series. *Radiology Case Reports* 2014;8(5):27-34

39. Wendlova J, **Freeman MD**. The Slovak Regression Model of Fall-Related Femoral Neck Fracture Risk. *Journal of Forensic Biomechanics* Vol. 4 2013), Article ID 235595, 5 pages doi:10.4303/jfb/235595

40. **Freeman MD**, Dobbertin K, Kohles SS, Uhrenholt L, Eriksson A. Serious head and neck injury as a predictor of occupant position in fatal rollover crashes. *Forensic Sci Int* 2012;222:228–33.

41. **Freeman MD,** Kohles SS. An examination of the threshold criteria for the evaluation of specific causation of mesothelioma following a history of significant exposure to chrysotile asbestos-containing brake dust, *Int J Occ Env Hlth* 2012;18(4):329-36.

42. **Freeman MD,** Fuerst M. Does the FDA have regulatory authority over adult autologous stem cell therapies? FDCA 21 CFR 1271 and the Emperor's New Clothes. *J Transl Med* 2012;10(1):60.

43. **Freeman MD,** Everson T, Kohles SS. Forensic epidemiologic and biomechanical analysis of a pelvic cavity blowout injury associated with ejection from a personal watercraft (jet-ski). *J Forens Sci* 2012 doi: 10.1111/j.1556-4029.2012.02250.x

44. **Freeman MD**, Kohles SS. Plasma levels of polychlorinated biphenyls, non-Hodgkin lymphoma, and causation. *J Environ Public Health* 2012;2012:258981. doi: 10.1155/2012/258981. Review.

45. Centeno CJ, Fuerst M, Faulkner SJ, **Freeman MD.** Is cosmetic platelet-rich plasma a drug to be regulated by the Food and Drug Administration? *J Cosm Derm* 2011;10:171–3.

46. Centeno CJ, Schultz JR, Cheever M, **Freeman M**, Faulkner S, Robinson S, Hanson R. Safety and Complications Reporting Update on the Re-Implantation of Culture-Expanded Mesenchymal Stem Cells Using Autologous Platelet Lysate Technique. *Cur Stem Cell Res & Ther* 2011;6(4):XX

47. **Freeman MD,** Kohles SS. Application of the Hill Criteria to the Causal Association of Post-Traumatic Headache and Assault. *Egypt J Forensic Sci* 2011;1:35-40.

48. **Freeman MD,** Kohles SS. Application of the Bradford-Hill Criteria for Assessing Specific Causation in Post-Traumatic Headache. *Brain Inj Prof* 2011;8(1):26-8.

49. **Freeman MD**, Kohles SS. An Evaluation of Applied Biomechanics as an adjunct to systematic specific causation in forensic medicine. *Wien Med Wochenschr* 2011;161:1-11.

50. Uhrenholt L, **Freeman MD**, Jurik AG, Jensen LJ, Gregersen M, Boel LW, Kohles SS, Thomsen AH. Esophageal injury in fatal rear-impact collisions. *Forensic Sci Int* 2011;206(1-3):e52-7.

51. **Freeman MD**. A Bayesian assessment of unexplained fracture as a forensic test of child abuse; quantification of uncertainty using the Error Odds approach. *Acta Medicinae Legalis et Socialis* 2010:179-84.

52. Nystrom NA, Champagne LP, **Freeman MD**, Blix E. Surgical fasciectomy of the trapezius muscle combined with neurolysis of the spinal accessory nerve; results and long-term follow-up in 30 consecutive cases of refractory chronic whiplash syndrome. *J Brachial Plexus and Peripheral Nerve Injury* 2010:5;7.

53. Centeno CJ, Schultz J, Cheever M, Robinson B, **Freeman MD**, Marasco W. Safety of autologous MSC transplantation: an in vivo MRI study of transplanted MSCs culture-expanded using a novel, platelet-lysate technique. *Cur Stem Cell Res & Ther* 2010;5:81-93.

54. Dagenais S, Gay RE, Tricco A, Mayer, JM, **Freeman MD**. North American Spine Society Contemporary Concepts in Spine Care: Spinal Manipulation Therapy for Acute Low Back Pain *Spine J* 2010 Oct;10(10):918-40.

55. Uhrenholt L, Schumacher B, **Freeman MD**. Road traffic fatalities in Aarhus Police District in 2000-2004 - medical investigations and legal consequences. *Ugeskr Laeger.* 2010 Sep 27;172(39):2683-2687. Danish

56. **Freeman MD**, Woodham M, Woodham A. The role of the lumbar multifidus in chronic low back pain; a review. *PM R* 2010 Feb;2(2):142-6.

57. **Freeman MD**, Centeno CJ, Kohles SS. A systematic approach to clinical determinations of causation in symptomatic spinal disc injury following motor vehicle crash trauma. *PM R* 2009;1(10):951-6.

58. **Freeman MD**, Rosa S, Harshfield D, Smith F, Bennett R, Centeno CJ, Kornel E, Nystrom A, Heffez D, Kohles SS. A case-control study of cerebellar tonsillar ectopia and head/neck (whiplash) trauma. *Brain Injury* 2010;24(7-8):988-94.

59. **Freeman MD**, Kohles SS. Applications and limitation of forensic biomechanics; a Bayesian perspective. *J Forensic Legal Med* 2010;17:67-77.

60. **Freeman MD**, Nystrom A, Centeno C, Hand M. Chronic whiplash and central sensitization; do a trigger points play an important role in pain modulation? *J Brachial Plex Peripher Nerve Inj* 2009 Apr 23;4:2.

61. **Freeman MD**, Hand ML, Rossignol AM. Applied Forensic Epidemiology: A Bayesian evaluation of forensic evidence in a vehicular homicide investigation. *J Forensic Legal Med* 2009;16(2):83-92.

62. Centeno CJ, Busse D, Kisiday J, Keohan C, **Freeman M**, Karli D Regeneration of meniscus cartilage in a knee treated with percutaneously implanted autologous mesenchymal stem cells. *Med Hypothese.* 2008 Dec;71(6):900-8.

63. Centeno CJ, Schultz J, **Freeman M**. Sclerotherapy of Baker's cyst with imaging confirmation of resolution. *Pain Physician* 2008 Mar-Apr;11(2):257-61.

64. Centeno CJ, Busse D, Kisiday J, Keohan C, **Freeman M**, Karli D. Increased knee cartilage volume in degenerative joint disease using percutaneously implanted, autologous mesenchymal stem cells. *Pain Physician* 2008 May;11(3):343-53.

65. **Freeman MD**, Centeno CJ. A fatal case of secondary gain; a cautionary tale. *Amer J Case Reports* 2008;9:97-103.

66. Centeno CJ, Elkins W, **Freeman M**, Elliott J, Sterling M, Katz E. Total Cervical Translation as a Function of Impact Vector as Measured by Flexion-Extension Radiography *Pain Physician* 2007 Sep;10(5):667-71.

67. **Freeman MD**, Rossignol AC, Hand M. Forensic Epidemiology: A systematic approach to probabilistic determinations in disputed matters. *J Forensic Legal Med* 2008;15(5):281-90.

68. Centeno CJ, Kisiday J, **Freeman MD**, Shultz JR. Partial regeneration of the human hip via autologous bone marrow nucleated cell transfer: a case study. *Pain Physician* 2006;9:135-7.

69. Croft AC, **Freeman MD**. Correlating crash severity with injury risk, injury severity, and long-term symptoms in low velocity motor vehicle collisions. *Med Sci Monit* 2005 Oct;11(10):RA316-21. Epub 2005 Sep 26.

70. **Freeman MD**, Croft AC, Nicodemus CN, Centeno CJ, Welkins WL. Significant spinal injury resulting from low-level accelerations: A case series of roller coaster injuries. *Arch Phys Med Rehab* November 2005;86:2126-30.

71. **Freeman MD**, Croft AC, Rossignol AC, Elkins W. Chronic neck pain and whiplash: a case/control study of the relationship between acute whiplash injuries and chronic neck pain. *Pain Res Manag* 2006;11(2):79-83.

72. Centeno CJ, **Freeman MD**, Welkins WL. A review of the literature refuting the concept of minor impact soft tissue injury. *Pain Res Manag* 2005;10(2):71-4.

73. Centeno C, Elliot J, Elkins W, **Freeman M**. A prospective case series of fluoroscopically guided cervical prolotherapy for instability with blinded pre and post radiographic reading. *Pain Physician* 2005;8(1):

74. Centeno CJ, Elkins WL, **Freeman M**. Waddell's signs revisited? *Spine* 2004 Jul 1;29(13):1392

75. **Freeman MD**, Nelson C. Injury Pattern Analysis as a means of driver identification *Laboratory Medicine* 2004;35(8):502-5.

76. **Freeman MD**, Olson D. Hemifacial tic following a low-speed motor vehicle crash. *J Whiplash Rel Dis* 2004;3(1).

77. **Freeman MD**, Nelson C. Injury pattern analysis as a means of driver identification in a vehicular homicide; a case study. *Forensic Examiner* Spring 2004;13(1):24-8.

78. Croft AC, Haneline MT, **Freeman MD**. Low speed frontal crashes and low speed rear crashes: is there a differential risk for injury? *Annu Proc Assoc Adv Automot Med. 2002;46:79-91.*

79. Croft AC, Herring P, **Freeman MD**, Haneline MT: The neck injury criterion (NIC): future considerations. *Accid Anal Prev* 2002;34(2):247-55.

80. **Freeman MD**, Croft AC, Rossignol AM. A critical evaluation of the methodology of a low back pain clinical trial *J Manipulative Physio Ther* 2000; 23(5):363-4.

81. **Freeman MD**, Croft AC, Rossignol AM, Weaver DS, Reiser M. A review and methodologic critique of the literature refuting whiplash syndrome. *Spine* 1999;24(1):86-98.

82. **Freeman MD**, Croft AC, Rossignol AM. Whiplash Associated Disorders (WAD) - Redefining Whiplash and its Management" by the Quebec Task Force: A Critical Evaluation. *Spine* 1998;23(9):1043-9.

83. **Freeman MD**, Fox DD, Richards TR. The superior intracapsular ligament of the sacroiliac joint: confirmation of Illi's ligament. *J Manipulative Physiol Ther* 1990;13(7):374-90.

Non-peer-reviewed publications

1. **Freeman MD**. The problem with probability. *Trial* March 2006, 58-61.

2. Croft AC, **Freeman MD**. Auto insurers and their new role in whiplash prevention—new rules, new risks, new tests. *Forum* 35(3):9-13, 2005.

3. O'Shanick G, Varney N, **Freeman MD**, et al. Blue Ribbon Panel Review of the Correlation Between Brain Injury and Roller Coaster Rides (Report to US Congress, Funded by the National Institute of Child Health and Human Development). February 25, 2003.

4. Croft AC, Herring P, **Freeman MD**, Centeno C, Haneline MT, Baric JJ: Late (chronic) whiplash injury. Public health perspectives amidst a controversial literature. JACA 40(8):26-32, 2003.

5. **Freeman MD**. Don't fall for defense fallacies. *Trial* 2000.

6. Seroussi R, **Freeman MD**. A review of original research by Brault et al. "Clinical response of human subjects to rear-end automobile collisions" *Injury Forum* 2000;2(5):49-50.

7. **Freeman MD**. The epidemiology of acute and chronic whiplash injury in the U.S. Proceedings of HWS-Distorsion (Schleudetrauma) & Leichte Traumatische, Hirnverletzung. Invaliditat und Berufliche Reintegration. Basel, Switzerland. June 29-30, 2000.

8. **Freeman MD**. Meta-analysis of whiplash prognosis studies. Proceedings of Whiplash 2000, Bath, England. May 16-18, 2000. pp 102-24.

9. **Freeman MD**, Croft AC, Reiser M. [reprint of A review and methodologic critique of the literature refuting whiplash syndrome] *Trial* March 1999

10. Croft AC, **Freeman MD**. From railway spine to whiplash. *Topics in Clinical Chiropractic* (Trauma) 1998;5(3):54-61.

11. **Freeman MD**, Croft AC, Reiser M. Die epidemiologie des Schleudertraumas - wo liegt die Schelle Zur Verletzung? (The epidemiology of whiplash - is there a reliable threshold for whiplash injury?) *HWS-Distortion (Schleudetrauma) & Leichte Traumatische, Hirnverletzung.* Edited by Ettlin TM and Mürner J. June 25-6, 1998:99-118.

12. **Freeman MD**. The first CIREN conference: motor vehicle crash-related trauma and biomechanical engineering. *JACA* 1998;35(4):54-61.

Theses

1. **Freeman MD.** *The Role of Forensic Epidemiology in Evidence-Based Forensic Medical Practice.* Thesis for completion of Doctor of Medicine degree, Umeå University. Responsible publisher under Swedish law: the Dean of the Medical Faculty of Umeå University, Printed by: Print och Media, Umeå, Sweden 2013. ISBN: 978-91-7459-729-5 ISSN: 0346-6612

2. **Freeman MD.** *A study of chronic neck pain and whiplash injuries.* Thesis for completion of Doctor of Philosophy degree, Oregon State University. *UMI Dissertation services*, Ann Arbor, MI. 1998:9820108.

3. **Freeman MD**. *Analysis of lumbar spinal strength using the doubly multivariate repeated measures design.* Thesis for completion of Master of Public Health degree, Oregon State University. 1995 (unpublished)

Books

1. **Freeman MD**, Zeegers M, Eds. Forensic Epidemiology; Principles and Practice. Elsevier, Amsterdam, NL. 2016

2. Nordhoff L, **Freeman MD**, Siegmund GP. Human Subject Crash Testing: Innovations and Advances. Society of Automotive Engineers, Detroit MI 2007

3. Berardinelli D, **Freeman MD**, DeShaw A. From Good Hands to Boxing Gloves; How Allstate Changed Casualty Insurance in America. Trial Guides 2008

4. **Freeman MD**. Litigating Major Auto Injury and Death Cases; Forensic Science (volume 2 of Litigating Major Auto Injury and Death Cases, Koehler K, Freeman MD). ThomsonWest:2006

5. **Freeman MD**. Litigating Minor Impact Soft Tissue Cases; Forensic Medicine (volume 2 of Litigating Minor Impact Soft Tissue Cases, Koehler K and Freeman MD). ThomsonWest 2001.

Book Chapters

1. **Freeman MD**. Cervical Sprain and Strain. Medscape. Updated April 2016. Available at: http://emedicine.medscape.com/.

2. **Freeman MD**, Franklin FA. Criminal Investigation. Chapter 15, in Forensic Epidemiology; Principles and Practice. Freeman M, Zeegers M, Eds. Elsevier, Amsterdam, NL. 2016.

3. **Freeman MD**, Franklin FA, Cahn P. Medical Negligence Investigation. Chapter 14, in Forensic Epidemiology; Principles and Practice. Freeman M, Zeegers M, Eds. Elsevier, Amsterdam, NL. 2016.

4. **Freeman MD**, Franklin FA. Consumer Product Defect Investigation. Chapter 13, in Forensic Epidemiology; Principles and Practice. Freeman M, Zeegers M, Eds. Elsevier, Amsterdam, NL. 2016.

5. **Freeman MD**. Motor Vehicle Defect Investigation. Chapter 12, in Forensic Epidemiology; Principles and Practice. Freeman M, Zeegers M, Eds. Elsevier, Amsterdam, NL. 2016.

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Scientific and Peer-reviewed Conference Proceedings and Abstracts

1. **Freeman MD**, Williams K, Eriksson A. Ballistic analysis of an attempted murder using a porcine model. *Proceedings of 70th Annual Meeting of the American Academy of Forensic Sciences* 2018 Feb 19-23: Seattle, WA. H130.

2. **Freeman MD**, Freeman EM. A probabilistic analysis of the cause of a traffic death following 2 crashes using national crash data. *Proceedings of 70th Annual Meeting of the American Academy of Forensic Sciences* 2018 Feb 19-23: Seattle, WA. H121.

3. **Freeman MD,** Lukasevic T, Williams K, Eriksson A. Characteristics of traffic crash related blunt traumatic aortic injury. *Proceedings of 68th Annual Meeting of the American Academy of Forensic Sciences* 2016 Feb 22-26: Las Vegas, NV H61:773-5.

4. **Freeman MD**. Concussion risk associated with head impact; an analysis of pooled data from helmeted sports. *Proceedings of the 12th Annual Conference of the North American Brain Injury Society* J Head Trauma Rehab 2015;30(3):E72-3.

5. Franklin F, **Freeman MD.** An analysis of the causal relationship between maternal/ prenatal cocaine use and stillbirth: results of a national hospital database study. *Proceedings of 67th Annual Meeting of the American Academy of Forensic Sciences* 2015 Feb 16-21: Orlando, FL H154:975-6.

6. **Freeman MD**. Biomechanical, Mechanical, and Epidemiologic Characteristics of Low Speed Rear Impact Collisions. *Proceedings of 67th Annual Meeting of the American Academy of Forensic Sciences* 2015 Feb 16-21: Orlando, FL. D11:517-8.

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23. Uhrenholt L, **Freeman M.** How microscopy can explain traffic crash-related cervical spine injury. 2011. Poster session presented at Årsmøde i Dansk Selskab for Retsmedicin og Dansk Selskab for Ulykkes- og Skadeforebyggelse [The Danish Traffic Medicine Society of the Danish Society for Forensic Medicine] November 3-5, 2011] Grenå, Denmark.

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31. **Freeman MD**. The Error Odds method of objectively assessing bioengineering based claims of causation; a Bayesian approach to test validity quantification (Special joint session of Jurisprudence and Engineering Sciences) *Proceedings of 62nd Annual Meeting of the American Academy of Forensic Sciences* Feb 2010, Seattle, Washington.

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38. **Freeman MD**. The Error Odds assessment of accuracy for tests in forensic medicine; a simple application of Bayes' Law. *XXI Congress of the International Academy of Legal Medicine* May 2009 Lisbon, Portugal

39. Uhrenholt L, Schumacher B, **Freeman MD**. A cross-sectional study of road traffic fatalities and vehicular homicide investigation practices in Denmark for 2000-2004. *Proceedings of 61st Annual Meeting of the American Academy of Forensic Sciences*. Feb 2009, Denver, Colorado.

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55. Johansson BH, **Freeman MD**. The prevalence of symptomatic cervical disc herniation in the Swedish population with asymptomatic degenerative disc disease (a cross-sectional study). *International Congress on Whiplash Associated Disorders* March, 2001. Berne, Switzerland.

56. **Freeman MD**, Centeno C, Croft AC, Nicodemus C. Significant spinal injuries resulting from low-level accelerations: a case series of roller coaster injuries. *Proceedings of Cervical Spine Research Society 28th Annual Meeting,* November 30-December 2, 2000:110-1.

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Scientific Commentary/Editorials/Letters

1. Uhrenholt L, Webb A, **Freeman MD**. Letter to the Editor regarding "Do X-ray-occult fractures play a role in chronic pain following a whiplash injury?" *Eur Spine J* DOI 10.1007/s00586-014-3362-3.

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SCIENTIFIC PRESENTATIONS

1. Freeman MD. Causation analysis in medical negligence. Radboud Summer School. Radboud Medical Center, August 14, 2017: Nijmegen, Netherlands.

2. Freeman MD. Injury causation analysis. Radboud Summer School. Radboud Medical Center, August 14, 2017: Nijmegen, Netherlands.

3. Freeman MD. Criminal applications of Forensic Epidemiology. Radboud Summer School. Radboud Medical Center, August 14, 2017: Nijmegen, Netherlands.

4. Freeman MD. Introduction to Forensic Epidemiology. Radboud Summer School. Radboud Medical Center, August 13, 2017: Nijmegen, Netherlands.

5. Freeman MD. Ballistic analysis of an attempted murder using a porcine model. *Proceedings of 70th Annual Meeting of the American Academy of Forensic Sciences* 2018 Feb 19-23: Seattle, WA.

6. Freeman MD. Evidence-based practice in Forensic Medicine; Principles of Forensic Epidemiology. Radboud Medical Center, October 9, 2017: Nijmegen, Netherlands.

7. Freeman MD. Incidence and risk factors for neonatal falls US Hospitals, 2003-2012. *Health Science Research,* Doernbecher Childrens' Hospital, Oregon Health & Science University, March 13, 2017, Portland, Oregon.

8. Freeman MD. Incidence and risk factors for neonatal falls US Hospitals, 2003-2012. *Research in Progress,* Department of Internal Medicine, Oregon Health & Science University School of Medicine, January 31, 2017, Portland, Oregon.

9. Freeman MD. Evidence-based practice in Forensic Medicine. Invited presentation to the Dutch National Forensic Institute (NFI). December 6, 2016 Maastricht University, Maastricht, Netherlands.

10. Freeman MD. Forensic Epidemiology: Principals & Practice Part 2: Investigation of specific causation. Gran Sesión de Epidemiología Forense. November 18, 2016 Universidad Libre, Cali, Colombia.

11. Freeman MD. Forensic Epidemiology: Principals & Practice Part 1: Investigation of specific causation. Gran Sesión de Epidemiología Forense. November 18, 2016 Universidad Libre, Cali, Colombia.

12. Freeman MD. Fatal crash investigation. World Reconstruction Exposition (WREX 2016). May 2-6, 2016. Orlando, Florida.

13. Freeman MD. Trends in police use-of-force related hospitalizations; an analysis of Nationwide Inpatient Sample data for 1998-2012. *Research in Progress,* Department of Internal Medicine, Oregon Health & Science University School of Medicine, November 10, 2015, Portland, Oregon.

14. Freeman MD. Concussion risk associated with head impact; an analysis of pooled data from helmeted sports. *12th Annual Conference of the North American Brain Injury Society*, April 29-May 1, 2015 San Antonio, Texas

15. Freeman MD. The role of risk in assessing cause in forensic investigation of injury and death. *American Medical Response biennial EMS training.* April 17, 2015, Mt. Hood, Oregon.

16. Freeman MD. Development of a pediatric fatal head trauma registry. *Research in Progress,* Department of Internal Medicine, Oregon Health & Science University School of Medicine, April 7, 2015, Portland, Oregon.

17. Freeman MD. Fatal crash investigation: methods and case presentations. Washington County CART Team training lecture. Tualatin Police Department, Tualatin, Oregon. March 4, 2015.

18. Freeman MD. An analysis of the causal relationship between maternal/ prenatal cocaine

use and stillbirth: results of a national hospital database study. *67th Annual Meeting of the American Academy of Forensic Sciences* 2015 Feb 16-21: Orlando, FL

19. Freeman MD. Biomechanical, Mechanical, and Epidemiologic Characteristics of Low Speed Rear Impact Collisions. *67th Annual Meeting of the American Academy of Forensic Sciences* 2015 Feb 16-21: Orlando, FL.

20. Freeman MD. Sexual abuse in the Boy Scouts: a preliminary analysis of Boy Scout ineligible volunteer files from 1945 to 2004. *Research in Progress,* Department of Sociology, Portland State University. December 18, 2014.

21. Freeman MD. Understanding chronic pain after whiplash trauma. *Lund University Hospital, Department of Rehabilitation Medicine.* December 11, 2014, Lund, Sweden.

22. Freeman MD. Forensic Applications of Epidemiology in Criminal and Civil Settings. *Richard Doll Building, Nuffield College, Oxford University.* December 10, 2014, Oxford, UK.

23. Freeman MD. The Efficacy of tPA in Preventing Long Term Poor Outcome After Ischemic Stroke: A Reanalysis of NINDS Data. *Research in Progress,* Department of Internal Medicine, Oregon Health & Science University School of Medicine, November 25, 2014, Portland, Oregon.

24. Freeman MD. Forensic Epidemiology and Bioterrorism. Full day course for public health and law enforcement. A joint training for public health, law enforcement, and emergency services. Sponsored by Charles County Department of Public Health and funded through a grant from the Centers for Disease Control and Prevention, Public Health Preparedness Cooperative Agreement. College of Southern Maryland. June 10, 2014. Waldorf, Maryland.

25. Freeman MD. Maternal cocaine exposure and still-birth risk. *Research in Progress,* Department of Internal Medicine, Oregon Health & Science University School of Medicine, May 20, 2014, Portland, Oregon.

26. Freeman MD. Forensic Applications of Epidemiology in Civil and Criminal Litigation. *9th International Conference on Forensic Inference and Statistics* August 19-22, 2014

27. Freeman MD. Investigation of a disputed mechanism of diffuse axonal injury following a low speed frontal crash. *65th Annual Meeting of the American Academy of Forensic Sciences,* Feb 21, 2014, Seattle, Washington.

28. Freeman MD. Public defense of dissertation for Doctor of Medicine degree, "The role of forensic epidemiology in evidence based forensic medical practice." *Section of Forensic Medicine, Department of Community Medicine and Rehabilitation, Faculty of Medicine, Umeå University.* November 6, 2013, Umeå, Sweden.

29. Freeman MD. Case studies in applied forensic epidemiology. Invited lecture, *University of Maastricht, Department of Complex Genetics and Epidemiology,* Maastricht, The Netherlands. October 31, 2013.

30. Freeman MD. The relationship between Chiari malformation, trauma, and chronic pain. *Karolinska Institute,* September 27, 2012, Stockholm, Sweden.

31. Freeman MD. Serious head and neck injury as a predictor of occupant position in fatal rollover crashes. *18th Nordic Conference on Forensic Medicine*, June 13-16, 2012 Aarhus Denmark.

32. Freeman M. Self-defense or attempted murder? A combined ballistic and traffic crash reconstruction of a Texas shooting. *18th Nordic Conference on Forensic Medicine*, June 13-16, 2012 Aarhus Denmark.

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34. Freeman MD. Forensic Epidemiologic Investigation of Traffic Crash-Related Homicide. *Årsmøde i Dansk Selskab for Retsmedicin og Dansk Selskab for Ulykkes- og Skadeforebyggelse* [The Danish Traffic Medicine Society of the Danish Society for Forensic Medicine] November 3-5, 2011] Grenå, Denmark.

35. Freeman MD. Traffic Crash Injuries 1960 to the present; how far we've come. Keynote address, *Årsmøde i Dansk Selskab for Retsmedicin og Dansk Selskab for Ulykkes- og Skadeforebyggelse* [The Danish Traffic Medicine Society of the Danish Society for Forensic Medicine] November 3-5, 2011] Grenå, Denmark.

36. Freeman MD. Is there a place for forensic biomechanics in evaluation of Probability of Causation? *8th International Conference on Forensic Inference and Statistics (ICFIS),* July 19-21, 2011; University of Washington, Seattle, Washington.

37. Freeman MD. Case studies in forensic epidemiology. *8th International Conference on Forensic Inference and Statistics (ICFIS)*, July 19-21, 2011; University of Washington, Seattle, Washington.

38. Freeman MD. The Error Odds method of objectively assessing bioengineering based claims of causation; a Bayesian approach to test validity quantification. Invited lecture; joint session of Jurisprudence and Engineering Sciences. *62nd Annual Meeting of the American Academy of Forensic Sciences* Feb 25, 2010, Seattle, Washington.

39. Freeman MD, Uhrenholt L, Newgard C. The effect of restraint use on skull vault fractures in rollover crashes. Engineering Sciences section, *62nd Annual Meeting of the American Academy of Forensic Sciences* Feb 26, 2010 Seattle, Washington.

40. Freeman MD, Uhrenholt L, Newgard C. Head injuries in lower speed collinear collisions; an analysis of the National Automotive Sampling System database. Engineering Sciences section, *62nd Annual Meeting of the American Academy of Forensic Sciences* Feb 26, 2010 Seattle, Washington.

41. Freeman MD. The Error Odds assessment of accuracy for tests in forensic medicine; a simple application of Bayes' Law. Invited presentation; *XXI Congress of the International Academy of Legal Medicine* May 2009, Lisbon, Portugal

42. Freeman MD. Forensic Epidemiology and Traumatic Brain Injury. Invited presentation; *VII World Congress on Brain Injury, International Brain Injury Association* April 2008 Lisbon, Portugal.

43. Freeman MD, Hand M. Bayesian analysis of predictive characteristics in suicidal versus homicidal hanging deaths: A case study in forensic epidemiology. *59th Annual Meeting of the American Academy of Forensic Sciences* February 19-24, 2007, San Antonio, Texas.

44. Freeman MD. Probability and pathologic findings in suicidal versus homicidal hanging deaths; a case study *16th Nordic Conference on Forensic Medicine* June 15, 2006, Turku, Finland.

45. Freeman MD. Injury Pattern Analysis as a means of driver determination in a vehicular homicide investigation *16th Nordic Conference on Forensic Medicine* June 16, 2006, Turku, Finland.

46. Freeman MD. Probability and pathologic findings in suicidal versus homicidal hangings; a case study. Grand Rounds *Institute of Forensic Medicine, Aarhus University, Aarhus, Denmark*. October 27, 2005.

47. Freeman MD. Road Traffic Crashes- mechanisms, injuries and analysis. Invited lecture (Keynote address) *Danish Society for Automotive Medicine* Aarhus, Denmark. October 27, 2005.

48. Freeman MD. The Defense Medical Evaluation: Issues, Ethics and Pitfalls. *2nd Annual International Whiplash Trauma Congress* Breckenridge, Colorado. February 26, 2005.

49. Freeman MD. Injury Pattern Analysis in Fatal Traffic Crash Investigation *American Academy of Forensic Sciences' 57th Annual Meeting* New Orleans, Louisiana. February 24, 2005.

50. Freeman MD. Independent Medical Evaluations and secondary gain. Grand Rounds, *Department of Psychiatry, Oregon Health & Science University School of Medicine* November 2, 2004.

51. Freeman MD. The epidemiology of crash-related trauma. Invited lecture. Grand Rounds *Peace Health Hospital* Longview, Washington. March 30, 2004.

52. Freeman MD. Injury pattern analysis: the practical application to the investigation of crash related death. Grand Rounds Department of Pathology, *Oregon Health Sciences University* Portland, Oregon. January 21, 2004.

53. Freeman MD. Literature critique, Whiplash Updates. Invited lecture. *British Columbia Chiropractic Association* Vancouver, British Columbia, Canada. October 23, 2003.

54. Freeman MD. Catastrophic crash cases and probability. Invited lecture. *Paris American Legal Institute* Florence, Italy. September 22, 2003.

55. Freeman MD. Injury pattern analysis as a means of driver identification in a vehicular homicide; a case study. *International Traffic Medicine Association Annual Meeting.* Budapest, Hungary. September 17, 2003.

56. Freeman MD. Fatal head injury crashes in a rural Oregon county, 1990-1999. *International Traffic Medicine Association Annual Meeting.* Budapest, Hungary. September 16, 2003.

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62. Freeman MD. Epidemiologic and medical aspects of whiplash injury. *Swedish Orthopedic Society* Stockholm, Sweden. May 17, 2002.

63. Freeman MD. Epidemiologic considerations of whiplash injuries. Invited lecture. *European Chiropractic Union Annual Congress* Oslo, Norway. May 9, 2002.

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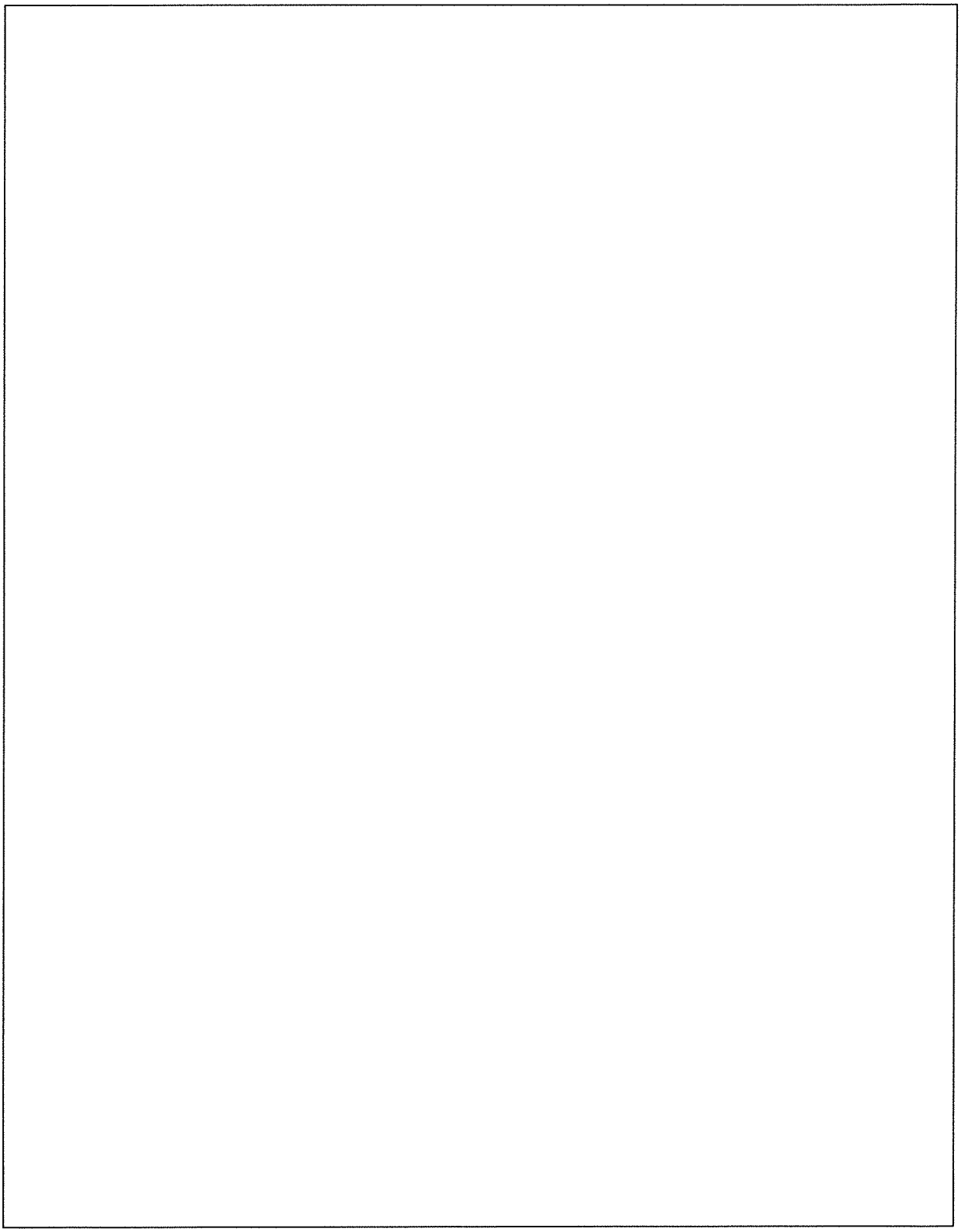

Erb/Duchenne's Palsy: A Consequence of Fetal Macrosomia and Method of Delivery

LYNNE V. McFARLAND, PhC, MAUREEN RASKIN, MS, JANET R. DALING, PhD, AND THOMAS J. BENEDETTI, MD

Methods of delivery, maternal and neonatal characteristics were examined to determine their role in the occurrence of Erb/Duchenne's palsy. Data from 210,947 Washington state birth certificates from 1980 through 1982 were examined. The incidence was 50.2 cases of Erb's palsy per 100,000 live births. A case control study design was used to analyze 106 cases and 386 controls by both univariable and multivariable analysis. Birth weight was shown to be a significant risk factor regardless of which method of delivery was used. A high birth weight infant (4001–4500 g) had 2.5 times the risk of incurring an upper brachial plexus injury compared with normal size infants (2501–4000 g). The risk for infants greater than 4500 g increased another tenfold (OR = 21.0). When birth weight was controlled for in the analysis, midforceps (OR = 18.3), vacuum extraction (OR = 17.2), and low forceps (OR = 3.7) remained significantly associated with the Erb's palsy. Delivery by cesarean section was associated with a significant protective effect (OR = 0.5) compared with instrumental vaginal delivery. These data demonstrate a high risk for serious birth injury associated with instrumental midpelvic delivery. ***(Obstet Gynecol 68:784, 1986)***

Injuries to the brachial plexus occur when there is a strong lateral traction on the head and neck or a downward traction on the shoulders, which causes adduction and internal rotation of the shoulder with pronation of the forearm. Damage may range from minimal to severe: from stretching of the nerve, to hemorrhage within the nerve, tearing of the nerve or root, or avulsion of the root with resulting cervical cord injury.[1] Differentiation of the types of injuries is based upon which cervical roots are involved. Erb/Duchenne's palsy is an upper brachial plexus injury involving cervical roots of C_5 to C_6. Associated unilateral paralysis of the diaphragm has also been reported.[2–4] Recent information suggests that about 70% of Erb's palsy patients recover by 3–13 months of age.[5,6] Klumpke's palsy, a lower brachial plexus type involving roots from C_7 to T_1, has a much poorer prognosis but is far less common than Erb's.[1]

Due to the low incidence of brachial plexus injuries, there has been a paucity of accurate data concerning true incidence as well as prognosis. The best estimates place the incidence between 35 and 57 per 100,000 live births, but such information is at least ten years old.[6,7] The authors felt there was a need for a population-based study to delineate those factors associated with upper brachial plexus injuries. The specific objectives of this study were 1) to identify which methods of delivery are associated with an increased risk of Erb's palsy while controlling for other variables, 2) to quantitate the risk associated with each of the methods, and 3) to examine birth weight as both an independent risk factor and a modifying variable.

Methods

A case-control design was used to examine data from 210,947 Washington state birth certificates from 1980 through 1982. All cases of Erb's palsy as coded on the birth certificates were identified. To assure the accuracy of the computer data to be used, patients' birth certificates were examined and compared with that coded on the computer tapes. Based on power considerations, a case-control ratio of 1:3 was used. Controls were randomly selected from those certificates where Erb's palsy was not indicated. Univariable and multivariable analyses were performed on the data obtained. Linked birth and infant death certificates provided data on mortality of Erb's palsy infants. Cause of death was listed by ICD codes.

Analysis of cesarean section was performed by pooling both primary and repeat cesarean sections. Be-

From the Department of Epidemiology, University of Washington; and the Department of Obstetrics and Gynecology, University Hospital, Seattle, Washington.

cause use of high forceps is a rare clinical event and only two categories of forceps use ("low" or "other") are present on the Washington birth certificates, "other" forceps was assumed to mean midforceps and, for purposes of presentation, was indicated as such. In addition to birth weight and method of delivery, the distributions among cases and controls for a number of other potentially significant factors were also examined. These variables included: attendant at birth, labor complications, neonatal sex, and maternal factors of age, race, parity, and diabetes status.

Data were analyzed by two methods. For the univariable analysis, a separate odds ratio and 95% Taylor series confidence interval were calculated for each variable. Odds ratios were significant at $P \leq .05$ when confidence intervals did not include one. Birth weight was classified as low (2500 g or less), normal (2501–4000 g), high (4001–4500 g), or very high (over 4500 g). The baseline comparisons used in calculating the crude odds ratios for the exposures were as follows: methods of delivery: unassisted vaginal delivery; attendant at birth: MD; number of prior pregnancies: none; maternal age: 20–35 years old; birth weight: normal (2501–4000 g); race: white; and neonatal sex: female. This method gave risk estimates for each of the exposures, but not independent of one another.

To account for collinearity of variables, adjust for simultaneous exposures, and reveal any interaction between these exposures, logistic regression was performed using the logistic regression program GLIM (General Linear Interactive Modeling, Royal Statistical Society, London, 1977). Due to the association of high and very high birth weight with Erb's palsy, birth weight was controlled for in the logistic model. Seven patients and 12 control subjects had more than one method of delivery listed (eg, other forceps and low forceps, other forceps and cesarean section, other forceps and vacuum extraction, or low forceps and vacuum extraction). In eight instances where induced labor was indicated, no method of delivery was identified. This resulted in the exclusion of eight subjects from analysis of method of delivery. Regression variables were fitted by a nested hierarchy approach. Coefficients (βs) of these regression variables were tested for significance using differences of log likelihood statistics interpreted as χ^2 as described by Breslow and Day.[8] Such coefficients can be interpreted as odds ratios (e^{β}), adjusted for other variables in the model. Ninety-five percent confidence intervals were calculated from the coefficients as $e^{\beta \pm 1.96\ (se\beta)}$. An omnibus test to assess goodness-of-fit of the final model was performed.

Table 1. Frequency Distribution of Selected Variables for Erb's Palsy Patients and Control Subjects

Variables	Erb's palsy	Controls
Method of delivery*		
Spontaneous	47 (42.3)	271 (69.1)
Cesarean section	4 (3.6)	46 (11.7)
Vacuum extraction	9 (8.1)	5 (1.2)
Low forceps	32 (28.8)	55 (14.0)
Midforceps	14 (12.6)	5 (1.2)
Total documented methods	106	382
Complications		
Asphyxia	6 (5.7)	2 (0.5)
Prolonged labor	5 (4.7)	16 (4.1)
Induced labor	4 (3.8)	8 (2.1)
Breech presentation	5 (4.5)	10 (2.5)
Attendant at birth†		
MD	93 (88.6)	361 (94.0)
Osteopath	6 (5.7)	8 (2.1)
Other non-MD	6 (5.7)	15 (3.9)
Maternal factors		
Diabetes	3 (2.8)	0
Age		
< 20 yr	27 (25.5)	64 (16.6)
> 35 yr	3 (2.8)	12 (3.1)
Race		
Black	9 (8.5)	13 (3.4)
White	90 (84.9)	341 (88.3)
Other	7 (6.6)	32 (8.3)
No. of prior pregnancies‡		
None	39 (37.9)	126 (32.6)
1–2	45 (43.7)	193 (50.0)
> 2	19 (18.4)	67 (17.4)
Neonatal factors		
Birth weight†		
Low (≤ 2500 g)	2 (1.9)	9 (2.3)
Normal (2501-4000 g)	52 (49.5)	320 (82.9)
High (4001–4500 g)	17 (16.2)	45 (11.6)
Very high (Over 4500 g)	33 (31.4)	12 (3.1)
Sex		
Male	61 (57.5)	197 (51.0)
Low Apgar score (≤ 7)§		
One minute	67 (65.7)	78 (20.3)
Five minutes	27 (26.5)	12 (3.1)

Number in parentheses denotes relative frequency.
* May be more than one category.
† One missing value (cases).
‡ Three missing values (cases).
§ Four missing values (cases).

Results

Among the 210,947 Washington state births from 1980 through 1982, 106 cases of Erb's palsy were clearly documented (an incidence of 50.2 cases per 100,000 live births).

The characteristics of case and control groups are shown in Table 1. Of interest are the appreciable differences between patients and control subjects for asphyxia, birth weight, low Apgar score, attendant at birth, and several methods of delivery.

Table 2. Crude Odds Ratios for Erb's Palsy

Variables	Odds ratio	95% Confidence intervals
Complications		
Asphyxia	11.6	(2.8, 47.8)
Prolonged labor	1.1	(0.4, 3.2)
Induced labor	1.8	(0.5, 6.3)
Maternal factors		
Age		
< 20 yr	1.6	(1.0, 2.8)
> 35 yr	1.0	(0.3, 3.6)
Race		
Nonwhite	1.4	(0.7, 2.5)
Parity		
1–2	0.8	(0.5, 1.2)
> 2	0.9	(0.6, 1.4)
Diabetes	∞	(0.8, ∞)*
Neonatal factors		
Sex		
Male	1.3	(0.8, 2.0)
Low Apgar score		
One minute	7.5	(4.6, 12.1)
Five minutes	11.2	(5.4, 23.0)

* Insufficient numbers to calculate OR. Confidence interval calculated by Fisher exact test.

Birth complications significantly associated with Erb's palsy included asphyxia (OR = 11.6), and low one- and five-minute Apgar scores (OR = 7.5 and OR = 11.2, respectively), as shown in Table 2. No significant association was seen between Erb's palsy and induced labor or with the maternal factors of race or parity. A trend towards significance was shown by a slightly elevated odds ratio (OR = 1.6) for maternal age under 20, but there was no association with maternal age over 35 years. Although there were slightly more male infants born with Erb's (OR = 1.3), the increase was not significant.

The results from logistic regression analysis are shown in Tables 3 and 4 and were similar to those obtained by univariable analysis. Neonatal birth weight remained the strongest predictor of Erb's palsy with an odds ratio of 2.4 for high birth weight (4001–4500 g) and OR = 21.0 for infants with a birth weight over 4500 g. Adjusting for birth weight allowed the determination of other factors, which may predict increased risk of Erb's palsy independent of birth weight. When compared with mechanically assisted deliveries (forceps or vacuum), significantly reduced risks were seen with cesarean section (OR = 0.1, 95% CI=0.02, 0.3) and unassisted spontaneous vaginal deliveries (OR = 0.2, 95% CI = 0.1, 0.3).

While the risk of Erb's in breech presentations was reduced from a univariable odds ratio of 11.7 to an adjusted odds ratio of 5.6 when the effects of birth weight and methods of delivery were controlled for, it nonetheless remained significantly elevated. Delivery by a non-MD (midwife, nurse, corpsman) showed an increased risk (OR = 3.1), which did not quite reach significance. On the other hand, delivery by an osteopath was associated with a significantly higher risk than delivery by an MD (OR = 4.9).

Table 3. Logistic Regression Analysis of Variables Associated With Erb's Palsy*

Variables	Odds ratio*	95% Confidence intervals
Birth weight		
Normal (2501–4000g)	1.00	
Low (≤ 2500 g)	2.4	(0.5, 10.4)
High (4001–4500 g)	2.4	(1.2, 4.8)
Very high (> 4500 g)	21.0	(9.5, 46.6)
Attendant at birth		
MD	1.0	
Osteopath	4.9	(1.5, 16.3)
Other non-MD	3.1	(1.0, 9.8)
Complications		
Breech presentation	5.6	(1.4, 22.7)

* Each variable is adjusted for others listed in table.

Specific methods of delivery were analyzed using unassisted spontaneous vaginal delivery as the baseline while controlling for birth weight and attendant at birth, as shown in Table 4. Use of low forceps was associated with increased risk (OR = 3.7). Use of midforceps and vacuum extraction resulted in even higher risk estimates (OR = 18.3 and OR = 17.2, respectively). Cesarean sections were protective for Erb's palsy (OR = 0.5). These results were not confounded by maternal age, parity, race, or neonatal sex. The risks associated with the methods of delivery were also not found to significantly vary across birth weight strata (no effect-modification). The inclusion of variables in Tables 3 and 4 (birth weight, attendant, methods of delivery, and breech presentation) in the logistic model was sufficient to predict the occurrence

Table 4. Logistic Regression Analysis of Methods of Delivery*

Method of delivery	Odds ratio	95% Confidence intervals
Unassisted vaginal	1.0	
Low forceps	3.7	(2.0, 7.0)
Midforceps	18.3	(5.7, 59.3)
Vacuum extraction	17.2	(5.1, 58.2)
Cesarean section	0.5	(0.1, 1.9)

* Controlled for birth weight, type of attendant, and breech presentation.

of Erb's palsy in our data (Omnibus test: $X_9^2 = 4.3$, $P > .80$).

Four of the 106 infants born with Erb's palsy died during the first year of life. The causes of death were identified by ICD codes as follows: 481 (pneumococcal pneumonia), 763.8 (other specified complications of labor and delivery), 769 (respiratory distress syndrome), and 798.0 (SIDS).

Discussion

Erb's palsy is usually a complication of shoulder dystocia, although it has been associated with complicated breech deliveries. Only four injured infants in this series were delivered in the breech position, one of which was an 830-g baby delivered by cesarean section. Therefore, the brachial plexus injuries in this study were nearly all the result of shoulder dystocia. A conservative estimate of the incidence of shoulder dystocia is 3 in 1000 vaginal births.[9] If that figure was used to estimate the total number of shoulder dystocia cases in the state during the time of our study, the rate of brachial plexus injury in those infants sustaining shoulder dystocia would be 1 in 6.

This study confirms the association of high birth weight with Erb palsy[6,8,10] and further refines the risks associated with fetal macrosomia and method of delivery. Use of logistic regression allowed for the assessment of birth weight as an independent variable as well as allowing for the calculation of other risk factors independent of birth weight. By this method we are able to show the highest risk occurs in infants weighing in excess of 4.5 kg (OR = 21.0). Further analysis of the data demonstrated the risk of brachial plexus injury to be 1 in 165 (33 of 5442) for any infant with birth weight over 4500 g. Data from all Washington state birth certificates showed an overall cesarean section rate of 25% in infants with extreme fetal macrosomia. Of these, 18% were primary cesarean sections, and 7% were repeat cesarean sections. If one eliminates the patients who had cesarean section from consideration of the population at risk, the incidence of brachial plexus injury increases to 1 in 124. Assuming that there is one brachial plexus injury for each six shoulder dystocia, the incidence of shoulder dystocia in infants weighing greater than 4.5 kg would be 1 in 20. This group accounted for 32% (33 of 104) of infants in this study suffering a brachial plexus injury. If a reliable method of predicting fetal weight becomes available in the near future, this group of infants would seem to be at such high risk for birth injury that cesarean delivery would seem justifiable on the basis of weight alone.

When one considers the risk to infants weighing between 4001 and 4500 g, the indication for primary cesarean section becomes less compelling. Seventeen infants in this group suffered brachial plexus injury. However, there were 23,523 infants delivered in this same period that were in this weight range, giving an incidence of 1 brachial plexus injury per 1383 births compared with an incidence of 1 in 3294 if the infant is less than 4000 g. Despite the increased risk of Erb's palsy associated with birth weight between 4001–4500 g (OR = 2.4), the high frequency (11.2%) of infants in this group does not justify a strategy of elective cesarean delivery on the basis of birth weight alone. Even if a reliable method of estimation of fetal weight is developed, a plan of cesarean delivery for infants over 4000 g would result in an excessive number of cesarean deliveries.

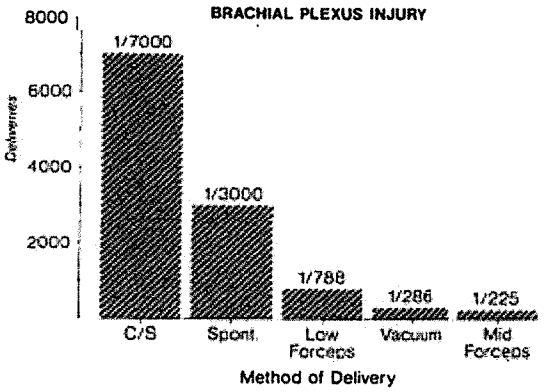

Figure 1. Incidence of Erb's palsy by method of delivery.

The highest risk factor that can be avoided with the present state of knowledge is the performance of midpelvic delivery. Midforceps delivery carried the highest risk for brachial plexus injury (OR = 18.3) even when corrected for birth weight. Figure 1 shows the incidence of Erb's associated with the different methods of delivery. Given the limitations of a study using birth certificates, we were not able to ascertain the reasons for the midforceps deliveries or the station from which they were performed. However, most clinicians have abandoned the use of forceps at 0 and +1 station and have limited midforceps procedures to station +2 and below. Furthermore, most clinicians do not list deliveries requiring minor degrees of rotation on the pelvic floor as midforceps procedures. It seems reasonable to infer from these data that midforceps procedures were those requiring significant amounts of rotation (over −90 degrees).

Vacuum extraction was also associated with a very high relative risk of Erb's palsy (OR = 17.2). Clinical information regarding vacuum extraction was not available. However, the usual practice is to use outlet forceps when the head is on the pelvic floor and

selection of other instruments when the criteria for low forceps are not satisfied. Many delivery attendants feel the use of the vacuum extractor offers advantages over use of forceps for both low and midpelvic deliveries. It has been reasoned that it is impossible to exert excessive traction with the vacuum extractor. According to this line of reasoning, when the traction forces exceed 30 pounds of pressure, the cup will separate from the head. In the absence of technical difficulties, this usually indicates the presence of cephalopelvic disproportion and cesarean delivery is indicated.

Unfortunately, the use of the vacuum extractor allows a delivery attendant with less technical skill and possibly less experience and judgement to attempt deliveries previously included in the midforceps category. Furthermore, the head with the vacuum extractor attached presents 8% less width than does the same fetal head during a forceps delivery. This fact may allow skilled technicians with the vacuum extractor to deliver very large infants without excessive force.

Our findings are consistent with the hypothesis that the vacuum extractor is being used instead of forceps for midpelvic deliveries. However, we cannot discount the possibility that these were low pelvic deliveries that benefited from the narrower diameter required to transverse the pelvis.

As expected, cesarean section offered a significant protective effect with regard to the development of brachial plexus injury in the infant. However, a finding that merits further explanation is the occurrence of brachial plexus injuries in four patients delivered by cesarean section. One infant presenting in the breech position weighed only 830 g. The three vertex presenting infants with Erb's palsy were not excessively large (2778, 3182, and 3997 g). In one instance fetal distress was listed as the reason for cesarean section delivery and in another case midforceps delivery was unsuccessful. From these data it would appear that brachial plexus injury may result from: 1) lateral traction during cesarean delivery, 2) antecedent events during deep engagement, and/or 3) attempted vaginal delivery.

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Address reprint requests to:
Lynne V. McFarland, PhC
School of Public Health & Community Medicine
Department of Epidemiology
SC-36
University of Washington
Seattle, WA 98195

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CLINICAL ARTICLE

Obstetrics

WILEY GYNECOLOGY OBSTETRICS

A multistate population-based analysis of linked maternal and neonatal discharge records to identify risk factors for neonatal brachial plexus injury

Michael D. Freeman[1,2,3*] | Shaun M. Goodyear[3] | Wendy M. Leith[3]

[1]Oregon Health and Science University School of Medicine, Portland, OR, USA

[2]CAPHRI School of Public Health and Primary Care, Maastricht University, Maastricht, Netherlands

[3]Forensic Research and Analysis, Portland, OR, USA

*Correspondence
Michael D. Freeman, CAPHRI School of Public Health and Primary Care, Maastricht University, Maastricht, Netherlands.
Email: forensictrauma@gmail.com

Abstract

Objective: To evaluate the interaction and contribution of maternal and fetal risk factors associated with neonatal brachial plexus injury (BPI).

Methods: In a case–control study, matched maternal and neonatal discharge records were accessed from US State Inpatient Databases for New Jersey (2010–2012), Michigan (2010–2011), and Hawaii (2010–2011). Univariate and multivariate logistic regressions were used to evaluate associations between risk factors and BPI. Area under the receiver operating characteristic curve was used to build predictive models, including two stratified models evaluating deliveries among obese and diabetic cohorts.

Results: Among 376 325 deliveries, BPI was diagnosed in 274 (0.1%). Significant BPI risk factors included maternal obesity (odds ratio [OR] 2.7, 95% confidence interval [CI] 1.7–4.4), maternal diabetes (OR 4.6, 95% CI 3.0–7.0), use of forceps (OR 4.6, 95% CI 2.3–9.0), and vacuum assistance (OR 2.3, 95% CI 1.7–3.3). After adjusting for shoulder dystocia and other predictive factors, cesarean reduced the risk of BPI by 88% (OR 0.1, 95% CI 0.07–0.2). When stratified by obesity and diabetes, the ORs for BPI increased significantly for macrosomia, forceps, and vacuum assistance.

Conclusion: The analysis confirms and quantifies more precisely the impact of risk factors for neonatal BPI, and provides a reliable basis for evidence-based clinical decision-making models.

KEYWORDS

Cesarean delivery; Maternal diabetes; Maternal obesity; Neonatal brachial plexus injury; Shoulder dystocia, State Inpatient Databases

1 | INTRODUCTION

Neonatal brachial plexus injury (BPI) results from excessive caudal traction at the fetal shoulder and spine that occurs during the second stage of labor, leading to a stretch or avulsion injury of the spinal nerves traveling from the cervical spine to the upper extremities.[1] The prevalence of BPI is approximately 1.5 per 1000 births and, although most injuries (approximately 70%) resolve within the first few months after birth, a substantial minority are permanent.[2]

The most common risk factor for BPI is shoulder dystocia, occurring in approximately 1.4% of all deliveries and 55% of BPI cases.[2] The risk of shoulder dystocia is highly correlated with fetal macrosomia and other conditions such as maternal diabetes and obesity.[3] Cesarean delivery is the most assured way to avoid BPI secondary to shoulder dystocia, and is recommended for macrosomic pregnancies complicated by diabetes.[4,5] The practice is not without controversy, however, and a commonly raised issue is the cost-to-benefit ratio of elective cesarean delivery for macrosomic pregnancy.[4–6] Another challenge is

that macrosomia is difficult to predict, leading to estimates that more than 1000 elective cesarean deliveries—costing $4–8 million—would be required to prevent one case of BPI.[4] By comparison, the lifetime costs for a case of BPI, excluding potential loss in productivity and earning capacity, is estimated at more than $1 million.[5] A more accurate model for predicting shoulder dystocia would substantially improve the number needed to treat (NNT) value of elective cesarean delivery as a means of preventing BPI, potentially improving prior estimates that were based on less precise models.

The aim of the present study was to examine risk factors associated with BPI via a unique inpatient database with linked neonatal and maternal records in order to provide a more accurate assessment of BPI risk factors in a large sample of hospital discharge data, and also evaluate whether a more precise predictive model of dystocia and BPI might more accurately define previously estimated NNT values for cesarean delivery.

2 | MATERIALS AND METHODS

The present case–control study was based on maternal and neonatal data from deliveries in three US states between January 1, 2010, and December 31, 2012, abstracted from the State Inpatient Databases (SID), maintained by the Healthcare Cost and Utilization Project of the Agency for Healthcare Research and Quality (HCUP). The SID is a de-identified database and as such, research based on SID data does not require informed consent or institutional board review.

The SID provides all inpatient discharge records for all hospitals across 46 states, and contains clinical and resource-use information found in a discharge abstract. Owing to privacy considerations, HCUP prohibits publishing details on cell sizes of 10 or smaller, and requires investigators to sign a data use agreement that governs the disclosure and use of these data.

Since 2010, New Jersey, Michigan, and Hawaii have provided a variable linking a mother's delivery record with her newborn's birth record, which provides a more comprehensive record of procedures performed during labor and other factors that may affect the newborn. For the present analysis, SID data for New Jersey (January 1, 2010, to December 31, 2012), Michigan (January 1, 2010, to December, 31, 2011), and Hawaii (January 1, 2010, to December 31, 2011) were queried for all births, with BPI as the outcome of interest. Predictive variables were based on previously published BPI risk or cofactors, including cesarean delivery, shoulder dystocia, breech presentation, macrosomia, multiple birth, maternal diabetes or obesity, induced labor, prolonged first and second stages of labor, forceps, vacuum extraction, manual assistance, and payer, as well as mother's ethnic origin (white vs non-white), age, and ZIP income quartile (a variable that estimates median household income quartile per patient's residential ZIP code). These factors were identified by International Classification of Diseases, Ninth Revision (ICD-9) codes (Table 1), or were defined by HCUP in the data.

All statistical analyses were performed with SAS version 9.4 (SAS Institute, Cary, NC, USA). To examine risk factors associated with BPI, crude odds ratios (ORs) and 95% confidence intervals (CIs) were calculated for each of the potential categorical risk factors. A Satterthwaite *t* test (for unequal variance) was used to assess the relationship between the continuous variable maternal age by BPI status.

TABLE 1 ICD-9 codes used.

Variable definition	ICD-9 code
Brachial plexus palsy	767.6
Shoulder dystocia	660.4x
Breech	72.5x 763.0
Macrosomia	Birth weight >4500 g, 766.0
Multiple birth	V31.xx-V37.xx
Maternal diabetes	Comorbidity indicator, 250.xx, 648.0x, 775.0
Maternal obesity	Comorbidity indicator
Induced labor	73.4, 73.01, 73.1
Prolonged first stage	662.0x
Prolonged second stage	662.2x
Forceps	72.0, 72.1, 72.2x, 72.3x, 72.4, 72.6, 73.3, 763.2
Vacuum extraction	72.7, 72.71, 72.79, 763.3
Manual-assisted delivery	73.5x
Cesarean	74.0, 74.1, 74.2, 74.4, 74.99, 763.4

Abbreviation: ICD-9, International Classification of Diseases, Ninth Revision.

Three predictive models were constructed using multivariate adjusted logistic regression to identify independent predictors of BPI among those found significant in the univariate analysis. The first model included all the data; the second and third models were stratified for deliveries by mothers with obesity or diabetes, respectively. The models were built using stepwise selection with an inclusion criterion of P=0.20 to enter the model and an exclusion criterion of P=0.05 to exit. A parallel process was used to construct a model for predicting shoulder dystocia among vaginal deliveries. The predictive ability of each model was assessed by using the area under the receiver operating characteristic (ROC) curve.

The NNT for a cesarean delivery to prevent one case of BPI was calculated by the adjusted OR, which was adjusted for potential confounders.[7] In some cases, cell sizes were too small to publish, as per the HCUP data use agreement, and only the ORs are presented. Unless otherwise specified, statistical significance was at a P value of 0.05 or less.

3 | RESULTS

Among a total of 376 325 deliveries, BPI was diagnosed in 274 (0.1%) newborns. Shoulder dystocia was observed in 3749 cases, of which 138 (3.7%) were associated with a diagnosis of BPI (OR 104.7, 95% CI 82.4–132.9). Macrosomia (birth weight >4500 g) was present in 3487 neonates, of whom 29 (0.8%) were diagnosed with BPI

(OR 12.8, 95% CI 8.7–18.8). Use of forceps and vacuum extraction were associated with increased risk of injury (OR 6.2, 95% CI 3.3–11.7, and OR 4.5, 95% CI 3.3–6.1, respectively). Maternal diabetes and obesity, defined as a body mass index (BMI, calculated as weight in kilograms divided by the square of height in meters) of 30 or higher, were also significantly associated with BPI (OR 7.4, 95% CI 5.1–10.7, and OR 3.2, 95% CI 2.1–4.9, respectively). Mother's ethnic origin (white vs non-white) and cesarean delivery were associated with decreased risk of BPI. Maternal age was approximately 1 year lower for newborns with BPI than for those without. The complete data are presented in Table 2.

After univariate analysis, a stepwise logistic regression model was constructed for all deliveries. Shoulder dystocia remained the strongest risk factor for BPI (OR 56.7, 95% CI 43.6–73.8), along with the use of forceps and vacuum extraction (OR 4.6, 95% CI 2.31–9.1, and OR 2.3, 95% CI 1.7–3.3, respectively). Maternal obesity and diabetes remained significant risk factors (OR 2.7, 95% CI 1.7–4.4, and OR 4.6, 95% CI 3.0–7.0, respectively). The complete data are presented in Table 3.

Among obese women (n=10 428), there were 4105 vaginal deliveries and 6323 cesareans. In this cohort, there were 23 (0.2%) cases of BPI. The variables in the final model included cesarean delivery, forceps, vacuum extraction, shoulder dystocia, prolonged second stage of labor, macrosomia, maternal diabetes, and mother's ethnic origin. Due to a lack of fit, the variable "payer" was removed from the final model. The complete data are shown in Table 4. The ROC area predicting BPI in the maternal obesity cohort was 0.88, indicating good predictive ability. On the basis of this analysis, it is estimated that, among obese pregnancies, 198 cesarean procedures would be required to prevent one case of BPI.

TABLE 2 Crude associations between neonatal brachial plexus injury and risk factors.[a]

Risk factor	All live-born neonates (n=376 325)	Live-born neonates with BPI (n=274)	Odds ratio (95% confidence interval)
Induced labor	72 407 (19.2)	81 (29.6)	1.8 (1.4–2.3)
Forceps	[b]	[b]	6.2 (3.3–11.7)
Vacuum extraction	18 393 (4.9)	51 (18.6)	4.5 (3.3–6.1)
Breech	[b]	[b]	2.1 (0.3–15.0)
Manual	145 207 (38.6)	117 (42.7)	1.2 (0.9–1.5)
Cesarean	142 670 (37.9)	15 (5.5)	0.1 (0.06–0. 2)
Dystocia	3749 (1.0)	138 (50.4)	104.7 (82.40–132.9)
Maternal diabetes	6617 (1.8)	32 (11.7)	7.4 (5.1–10.7)
Multiple pregnancy	[b]	[b]	0.4 (0.2–1.1)
Prolonged 1st stage	347 (0.1)	0	–
Prolonged 2nd stage	[b]	[b]	7.4 (3.5–15.7)
Macrosomia	3487 (0.9)	29 (10.6)	12.8 (8.7–18.8)
Maternal obesity	10 428 (2.8)	23 (8.4)	3.2 (2.1–4.9)
White ethnic origin	179 505 (47.7)	95 (34.7)	0.6 (0.5–0.8)
Payer			
Private insurance	231 306 (61.5)	117 (42.7)	Ref.
Self-pay	16 730 (4.5)	22 (8.0)	2.6 (1.7–4.1)
Medicare/Medicaid	122 086 (32.4)	131 (47.8)	2.1 (1.7–2.7)
Other	[b]	[b]	1.3 (0.5–3.5)
ZIP income quartile			
1	59 038 (15.7)	63 (23.0)	2.0 (1.4–2.8)
2	70 062 (18.6)	60 (21.9)	1.6 (1.2–2.2)
3	87 267 (23.2)	66 (24.1)	1.4 (1.0–2.0)
4	156 697 (41.6)	84 (30.7)	Ref.
State			
Hawaii	31 359 (6.9)	19 (6.9)	0.9 (0.5–1.4)
Michigan	68 250 (22.3)	61 (22.3)	1.3 (1.0–1.7)
New Jersey	276 716 (70.8)	194 (70.8)	Ref.

[a]Values are given as number (percentage) unless indicated otherwise.
[b]Number too small for publication as per the Healthcare Cost and Utilization Project data use agreement.

TABLE 3 Adjusted odds ratios for neonatal brachial plexus injury among all deliveries.[a]

Risk factor	Odds ratio (95% confidence interval)
Cesarean	0.1 (0.1–0.2)
Forceps	4.6 (2.3–9.0)
Vacuum extraction	2.3 (1.7–3.3)
Breech	8.0 (1.1–57.6)
Manual	0.7 (0.5–0.9)
Dystocia	56.7 (43.6–73.8)
Prolonged 2nd stage	2.6 (1.1–6.1)
Macrosomia	4.7 (3.0–7.3)
Maternal obesity	2.7 (1.7–4.4)
Maternal diabetes	4.6 (3.0–7.0)
White ethnic origin	0.6 (0.4–0.7)
Payer	
Private insurance	Ref.
Self-pay	2.0 (1.2–3.3)
Medicare/Medicaid	1.8 (1.3–2.3)
Other	1.2 (0.4–3.2)
State	
Hawaii	0.5 (0.3–0.9)
Michigan	1.0 (0.7–1.3)
New Jersey	Ref.

[a]Stepwise selection criteria: entry, P=0.2; exit, P=0.05.

Among the mothers with diabetes (n=6617), there were 2265 vaginal deliveries and 4352 cesarean deliveries. In this cohort, there were 32 (0.5%) cases of BPI. Stepwise selection resulted in a logistic model that included cesarean delivery, forceps, vacuum extraction, shoulder dystocia, macrosomia, and maternal obesity. The adjusted ORs are shown in Table 5. Analysis of the area under the ROC curve indicated good predictive ability of the model (ROC area 0.91). On the basis of this analysis in the diabetic cohort, an estimated 92 cesarean procedures would prevent one case of BPI.

The above results demonstrate that, even after adjustment for significant confounders, shoulder dystocia remains the single greatest risk factor for BPI. Therefore, potential risk factors for shoulder dystocia were investigated among vaginal deliveries. Stepwise logistic regression analysis was performed on the cohort of vaginal deliveries, and the variables included in the final model are shown in Table 6. After adjustment, the three greatest risk factors for shoulder dystocia were macrosomia, maternal diabetes, and vacuum extraction.

4 | DISCUSSION

The present analysis confirms other investigators' findings regarding the increased risk of BPI among pregnant women with obesity or diabetes, and the significant protective effect that cesarean delivery provides against BPI. The data showed that, for mothers with diabetes, 92 cesarean deliveries would be required to prevent one case of BPI. When applied to obese mothers, an NNT of 198 cesarean deliveries would be needed to prevent one case of BPI.

TABLE 4 Adjusted odds ratios for neonatal brachial plexus injury among obese mothers.[a]

Risk factor	Odds ratio (95% confidence interval)
Cesarean delivery	0.04 (0.01–0.31)
Forceps	9.7 (1.5–62.2)
Vacuum extraction	6.5 (1.9–21.7)
Dystocia	40.5 (15.5–105.8)
Prolonged second stage	39.6 (3.4–457.4)
Macrosomia	10.3 (2.4–45.0)
Maternal diabetes	6.6 (2.4–17.9)
White ethnic origin	0.3 (0.1–0.9)

[a]Stepwise selection criteria: entry, P=0.2; exit, P=0.05.

The precise number of cesarean deliveries needed to prevent a permanent case of BPI is less clear. A review of the literature indicated that obstetrician-led studies demonstrate a BPI permanency rate of 10%–17%, whereas studies conducted by pediatric and orthopedic surgeons report a higher proportion of persisting injury, ranging from 27% to 58%.[8] The wide variation in the reported rate of BPI permanency is attributable to inconsistencies in study methods: only some studies provide comprehensive criteria for evaluating persistent BPI, and others do not include expert consultation or adequate follow-up times.[8,9]

A sensitivity analysis applying the upper and lower bounds of previous permanency rates (10%–58%) to the present data indicated that a range of 159–920 cesarean deliveries would be needed to prevent one permanent case of BPI among diabetic pregnancies. Thus, at minimum, the present study enables the previously published NNT range of 91–1494 for diabetic pregnancies to be narrowed.[4,8,10] The cesarean NNT for permanent BPI injury among obese pregnancies spans a wider range; applying the same permanent BPI rates as above resulted in a cesarean delivery NNT of 341–1980. It is likely that the narrower range of NNT values observed in the present study, relative to previous estimates, is a result of the large study population (approximately 380 000 births) accessed via the SID data.

On the basis of estimated average total Medicaid-related charges for maternal and newborn care of $9131 and $13 590 for vaginal and cesarean deliveries, respectively, the increased cost of preventive cesarean procedures would range from $709 000 to $4.1 million per case of BPI prevented among pregnant women with diabetes. Applying the same analysis to the NNT range associated with pregnant women with obesity, the increased cost of preventive cesarean deliveries would range from $1.5 million to $8.8 million per case of BPI prevented.[11]

Independent or sequential use of forceps and vacuum extractor greatly increases the risk of BPI. Moreover, neonates requiring cesarean delivery after failed instrument-assisted delivery are more likely to incur trauma than are those who receive an immediate cesarean.[12] Similar to previous reports,[10,13,14] the present study found that forceps

TABLE 5 Adjusted odds ratios for neonatal brachial plexus injury among mothers with diabetes.[a]

Risk factor	Odds ratio (95% confidence interval)
Cesarean delivery	0.13 (0.03–0.46)
Forceps	12.1 (1.3–111.2)
Vacuum extraction	3.8 (1.2–11.0)
Dystocia	41.3 (17.6–96.8)
Macrosomia	4.2 (1.5–11.5)
Maternal obesity	3.5 (1.4–8.3)

[a]Stepwise selection criteria: entry, P=0.2; exit, P=0.05.

TABLE 6 Adjusted odds ratios for shoulder dystocia.[a]

Risk factor	Odds ratio (95% confidence interval)
Induction	1.5 (1.4–1.6)
Vacuum extraction	2.4 (2.1–2.6)
Prolonged second stage	1.6 (1.1–2.4)
Macrosomia	9.5 (8.1–11.2)
Multiple pregnancy	0.05 (0.01–0.19)
Maternal obesity	1.7 (1.4–2.1)
Maternal diabetes	3.6 (3.0–4.3)
White	1.5 (1.4–1.6)
Payer	
Private insurance	Ref.
Self-pay	0.9 (0.8–1.1)
Medicare/medicaid	1.1 (1.00–1.2)
Other	1.4 (1.1–1.8)
ZIP income quartile	
1	1.07 (0.96–1.20)
2	1.2 (1.1–1.4)
3	1.2 (1.1–1.3)
4	Ref.

[a]Stepwise selection criteria: entry, P=0.2; exit, P=0.05.

and vacuum extraction increased the risk of BPI among both obese and diabetic pregnancies. Additionally, predictive modeling indicated that vacuum extraction, but not forceps, is an independent risk factor for shoulder dystocia. The reason for this finding is not completely clear; however, others have suggested that the direction of force applied to the fetal head with vacuum extraction could increase impaction of the anterior fetal shoulder against the maternal pubic symphysis.[13] Alternatively, vacuum extraction is more likely to be used for deliveries in which fetal size tends toward the upper range and which are thus more prone to dystocia.[13] The statistical modeling in the present analysis was adjusted for fetal size and other factors predictive of dystocia; thus, the increased risk of BPI with vacuum extraction is more probably representative of a real association, rather than the result of a confounded relationship.

The strength of the present analysis lies in use of the SID, an annual inpatient database encompassing approximately 90% of all hospital discharges per state. By comparison, the Kids' Inpatient Database (KID) is a stratified sample of pediatric discharges derived from each SID, and is only available every 3 years. Most critically, the KID contains coding data that are only related to diagnoses and procedures associated with the newborn's birth record, and lacks information from the mother's discharge record. By contrast, maternal and neonatal records in selected SID datasets are linked. The difference in the information contained in the two datasets is exemplified by use of the ICD-9 code for forceps or vacuum extractor, which is denoted on the mother's record as a procedure, but documented in the newborn's record as a diagnosis only if use of the instrument resulted in an injury. In a prior study using KID data, Foad et al.[15] reported that 0.08% and 0.2% of newborns underwent forceps or vacuum extraction, respectively. In the present study, the respective rates were 1.7% and 6.1%. Thus, the KID does not contain documentation regarding more than 95% of instrumented deliveries, resulting in a probable source of bias.

Another source of bias associated with coding differences between the KID and SID is the use of ICD-9 code 763.1 in the former database. Along with shoulder dystocia, this code includes a vague description of "other malpresentation, malposition, and disproportion during labor and delivery," and may explain the finding of Foad et al.[15] of 100.9 times increased odds of BPI when "shoulder dystocia" is present, as compared with the increased odds of 56.7 (95% CI 43.6-73.8) found in the present study using a maternal diagnostic code specific to shoulder dystocia (660.4x).[15] Overall, the inability to adjust for both maternal and neonatal risk factors biased Foad et al.'s[15] estimates of association, and explains why an identifiable risk factor for BPI was reported in only 46% of their KID BPI cases, whereas at least one dichotomous risk factor was observed in 78% of SID BPI cases in the present study.

The present study has some limitations. Extrapolating the results to the whole US population is potentially limited by the analysis of data from only New Jersey, Michigan, and Hawaii. The limited geographic scope of the data and small number of BPI cases (in comparison with the KID) could affect the accuracy of the estimates owing to the large variation in the data. For example, Foad et al.[15] reported shoulder dystocia in 17.7% of neonates with BPI, whereas this condition was observed in 50% of cases of BPI in the SID data. This difference is likely attributable to the discrepancy in how dystocia is coded; however, both frequencies are within the range reported in the literature (26%–85%).[16–19] Further limitations of the analysis were the lack of information regarding the severity and duration of BPI (i.e. stretch vs avulsion, temporary vs permanent injury), and the inability to comprehensively stratify characteristics such as fetal size and maternal BMI. Nevertheless, the SID provides a useful resource to investigate the role of both maternal and fetal factors on newborn injury outcomes.

As the prevalence of maternal obesity and gestational diabetes rises, obstetricians and midwives are increasingly challenged with high-risk pregnancies and the need to reduce the risk of negative outcomes for both the mother and newborn.[20,21] The findings of the present study of an increased risk of BPI for instrument-assisted deliveries

among obese and diabetic mothers suggests that extra caution should be taken in managing such deliveries.

AUTHOR CONTRIBUTIONS

MDF and WML conceived and designed the study, and performed statistical analysis. All authors acquired, analyzed, and interpreted the data. MDF and SMG drafted the manuscript, and all authors critically revised it. MDF and SMG provided administrative, technical, and material support.

CONFLICT OF INTEREST

MDF provides consultation in medicolegal matters, including cases of BPI allegedly associated with birth trauma. The authors have no other conflicts of interest.

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